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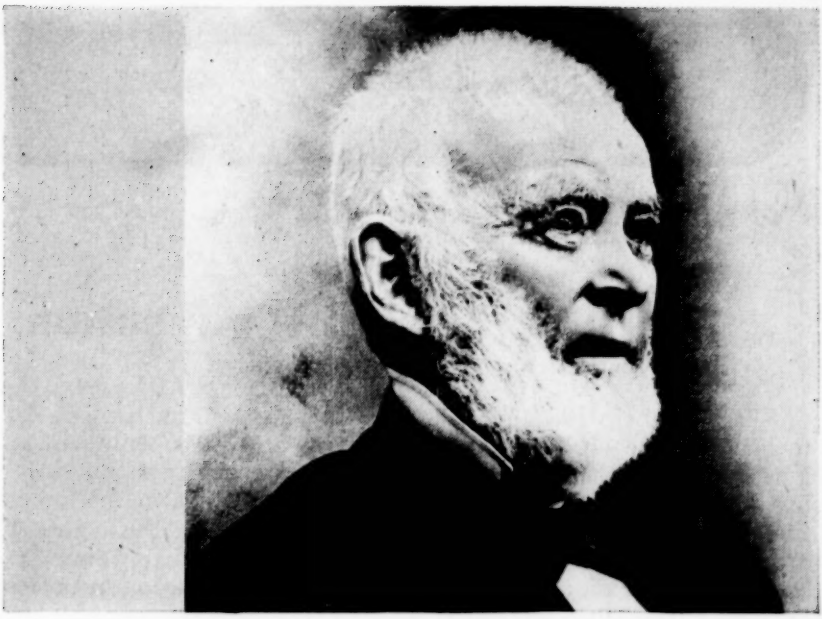

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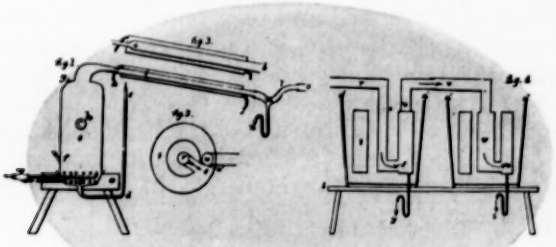

Diagrams for Early Ether Still from "Apparatus for the Preparation of Ether by Steam" Edward R. Squibb, M.D., The Journal of American Pharmacy, Sept. 1856.

Opinion Review

The 1952 Biennial Convention

The historic 1952 Biennial Nursing Convention in June marked the end of one era in organized nursing and the opening of another. Nurses differ about the effects of the new structure of nursing organizations on nursing and nursing service. Some see in the adoption of the two organization plan the realization of the highest potential for organized nursing. Others see in the allocation of power in, and the stated purposes of, the rebuilt A.N.A. the disintegration of professional nursing within that structure. The facts are these:

The two organization plan for national nursing organizations was approved by all the participating associations of nurses with the outstanding exception of the American Association of Industrial Nurses. The A.A.I.N. in April refused to lose its autonomy by dissolving to amalgamate with the new National League for Nursing. Organizations that did undergo dissolution were the National League for Nursing Education, the Association of Collegiate Schools of Nursing, and the National Organization for Public Health Nursing. (The National Association for Colored Graduate Nurses was previously absorbed by the A.N.A.)

The two organizations are (1) a slightly but significantly reconstructed A.N.A. and (2) a National League for Nursing built on the retained charter of the National League for Nursing Education. The revamped A.N.A. has the stated purposes of fostering high standards of nurse practice and of promoting the general welfare of nurses. The new N.L.N. has the stated purposes of fostering the development and improvement of nursing services and education.

The principal amendments to the A.N.A. bylaws as adopted at the recent Biennial indicate the course being charted by that organization. Of equal significance are amendments that failed to be approved. A major addition was the inclusion of a statement of functions of the association:

"1. To define functions of nurses and promote standards of professional nurse practice.

"2. To define qualifications for the practitioners of nursing, including those in the various nursing specialties.

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"3. To promote legislation and to speak for nurses in regard to legislative action concerning general health and welfare programs.
"4. To survey periodically the nurse resources of the nation.
"5. To promote and protect the economic and general welfare of nurses.
"6. To provide professional counseling service to individual nurses, and to their employers in regard to employment opportunities and available personnel.
"7. To cooperate with the National League for Nursing in activities which concern both organizations.
"8. To represent nurses and serve as their national spokesman with allied professional and governmental groups and with the public.
"9. To serve as the official representative of American nurses in the International Council of Nurses."

Two motions lost in relation to this section of the bylaws were: (1) to delete the entire section and (2) to amend the section to include as a function "To promote nursing services to patients in sickness and in health."

Of particular interest to nurses in the clinical nursing specialties was the amended bylaw pertaining to the formation of sections within the A.N.A.

"(b) A section shall be established for each of the following groups of professional nurses:
"(1) private duty nurses
"(2) general duty nurses
"(3) public health nurses
"(4) institutional nursing service administrators
"(5) educational administrators, consultants, and teachers
"(6) industrial nurses
"(7) special groups
"(c) A new national section, in addition to the seven named, may be established by the board of directors of this association if at least one-third of the state nurses associations have such a section.
"(d) Members of an occupational group in a state shall be eligible to apply for status as a section when they meet the following criteria:
"(1) In a state nurses association with a membership of 1500 or more, if they comprise at least 2½ per cent of that membership; or
"(2) In a state nurses association with a membership of less than 1500, if they number at least thirty-five; and after one of the two above named criteria has been met
"(3) If the members of the occupational group desiring status as a section have functioned as a conference group

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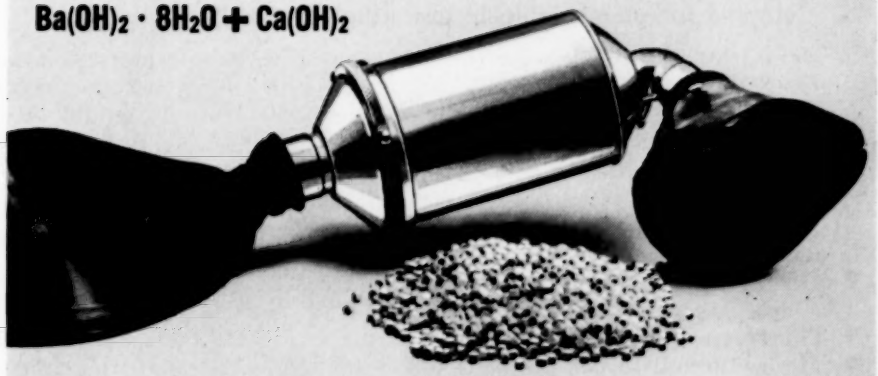

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within one of the established sections for a year; and

"(4) If they have demonstrated group interest; and

"(5) If they have developed a program; and

"(6) If the needs of such a group cannot be met in any existing section or by continuation as a conference group.

"(e) Membership in the Special Group Section of the American Nurses' Association and of the state nurses associations shall be open only to those nurses who are not eligible for membership in any other section."

The lack of provision for the numerous clinical nursing specialties —presumably, according to a comment of the Chair, because representation on the Board of Directors of clinical sections would mean narrow rather than organizational representation—was hotly and unsuccessfully contested by the psychiatric nurses. A motion to include a section for psychiatric nurses was lost.

The delineation of sections in the new organization of the A.N.A. is doubly significant in the light of:

- The change from state to sectional representation in the House of Delegates
- The retention of the sectional chairmen on the Board of Directors
- The reduction in the proportional number of delegates from one to every one hundred members to one for every two hundred members or fraction thereof.

An amendment that each state should have three delegates at large in addition to sectional representation was passed.

The House of Delegates at the 1952 Biennial also approved

- An increase in annual dues for active members from three to five dollars, despite protests that a decrease in membership would result
- The establishment of an executive committee composed of the president, three vice presidents, secretary, and treasurer, a coordinating council to work with the National League for Nursing, and a student council.

Other highlights of the A.N.A. House of Delegates meetings were the adoption of a resolution for a forty hour week, with two consecutive days off, for all nurses and the empowering of the Board of Directors to support legislation for selective service for nurses in the event of a National emergency.

The second of the two organizations, the National League for Nursing, held its organizational meeting on June 20, at which time Ruth Sleeper, R.N., director of the Massachusetts General Hospital School of Nursing, was elected the first president by the Board of Directors. The new N.L.N. has two divisions, one for nursing education with subdivisions for baccalaureate and diploma programs, and the other for nursing service, with subdivisions for hospital service, public health, and possibly industrial nursing. The broad base of membership includes all professional nurses, nonnurses involved or interested in nursing service or education, student nurses, and agencies

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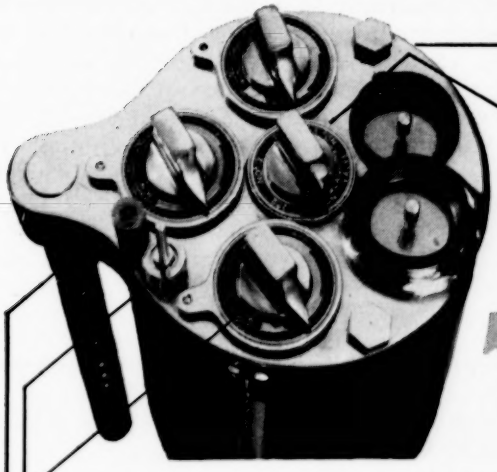

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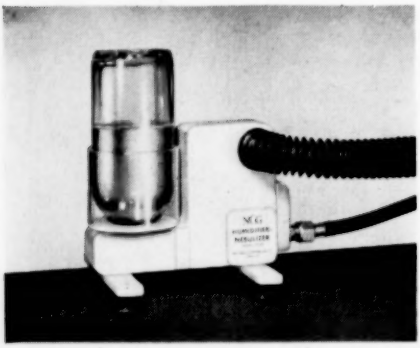
engaged in providing nursing service or education, with the possible inclusion in the future of practical nurses. In addition to its general aims of promoting the improvement and extension of nursing service and education, it is the N.L.N. that will act as the accrediting agency for educational programs in nursing.

The decisions made at the 1952 Biennial were apparently made with the full knowledge and consent of the members in the state and territorial affiliates. The decisions cannot help but affect not only professional nurses but also other workers in health programs, medical service agencies, and most important that subjective entity, the patient, who in the language of organized nursing has come to be known as the "consumer." An exciting future could be in store for the new N.L.N. as an effective agency for bringing about much needed improvements in nursing service and education. The future of the A.N.A. could be open to question in the light of the introduction to the report of the special committee on employment conditions for registered nurses. In accepting this report the A.N.A. House of Delegates went on record as subscribing to the thesis that it is the responsibility of hospital administrators to solve the nursing shortage and of the public to determine the type of nursing care that it wants and pay for it.



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The Journal OF THE
American Association of Nurse Anesthetists
Vol. XX No. 3 August, 1952

School Directors' Exchange

The year 1952 is an important one in the history of the American Association of Nurse Anesthetists, as it was on January 19 of this year that the accreditation program was accepted by the Board of Trustees. Every member has a right to feel a sense of pride in the organization because the adoption of a standard of education raises the professional level of the entire group and makes our future more secure.

Those who receive the most direct benefit from the accreditation program are the schools and the students. The conferences of the directors of schools and the Advisory to the Approval Committee accomplished more than the formulation of the criteria for accreditation. They brought forcibly to the attention of the directors that the problems of one are the problems of all, and that the sharing of them is a long step toward their solution. While the conferences were held for the purpose of reaching decisions regarding accreditation, many of the directors felt that they afforded a great deal of stimulation and assistance, and expressed the wish that more such meetings could be held. But conferences are too costly in money and time to be held as frequently as both stimulation and assistance are needed. And so it was suggested that an interchange of ideas might be effected through our publications.

This idea was not new, but in the past when the suggestion was made it was felt that school affairs were not of sufficient general interest to warrant a special section in either the JOURNAL or the A.A.N.A. NEWS BULLETIN. Now a better plan has been offered. The pilot committee, which drew up the recommendations for accreditation, included the suggestion that the American Association of Nurse Anesthetists sponsor a mimeographed publication to be called the *School Directors' Exchange*. The Board accepted the recommendation, and the directors will now have at their disposal a means of communication with each other. This publication will be exactly what the name

suggests, an exchange between the directors—a round robin letter, if you please. There will be no regular publication dates. When sufficient material has been sent to the Executive Office, it will be mimeographed, and each director will be sent a copy. The expense of this undertaking is insignificant, but the possibilities are very great. It can be a continuous conference of the directors in which problems can be presented, discussed, and perhaps solved. Questions can be asked and answered, helpful suggestions relayed, and useful articles reprinted. It will be of value to the exact extent to which it is appreciated and used by the directors. Because it will be informal and will reach only those who are engaged in the same endeavor, there need be no laborious phrasing of ideas or burdensome deadlines to meet. A letter to the *Exchange* will be like a letter to a friend.

The directors have long felt the need of a greater community of effort and have appreciated greatly the stimulation and assistance of every meeting with each other. The *School Directors' Exchange* can furnish refreshment in those arid periods between the meetings.—**Opal M. Schram, R.N.,** Chairman, Publications Committee.

Call to the Convention

As provided for in the Bylaws of this association, and at the direction of Verna E. Bean, president, we hereby issue this official call to the members of the annual meeting to be held in Philadelphia, September 15-18, 1952, at Convention Hall. The annual business session will be held on Tuesday, September 16.

Accomplished at the Executive Offices, 116 S. Michigan Ave., Chicago 3, Ill., this 10th day of June 1952.

(Signed) Florence A. McQuillen, R.N.
Executive Director

Neurologic Aspects of Hypoxia

Jules D. Levin, M.D. *
Milwaukee

All anesthetists are conversant with the common technic of controlled oxygenation in the closed method of anesthesia. In this article I want to discuss the equally common problem of what happens when controlled oxygenation is inadvertently transformed into a controlled oxygen want, that is, the problem of hypoxia. The actual symptoms and signs of oxygen want are well known, and I will only mention them in passing in due recognition of their importance. What I want to emphasize are the neurologic implications of oxygen want in degrees less severe than those causing death and, more especially, oxygen want of such range and severity as frequently leaves the patient without immediate, obvious neurologic residuals but only with an insensible neuronal loss.

Progress in anesthesia brought with it the closed method of anesthesia, and the problem of oxygen want, or hypoxia, has become of infinitely greater significance than was previously true. In the open technic of anesthesia the patient inhaled directly from and exhaled directly into the atmosphere. However, with the closed system of anesthesia the amounts of anesthetic agent and the amounts of oxygen received by the patient are under the absolute control of the anesthetist. Hence, oxygenation during anesthesia is left entirely to the good judgment and alertness of the anesthetist. With this realization of the importance of the anesthetist as part of the surgical team, let us briefly review some of the basic concepts in the physiology of anesthesia before we pass to the problem of oxygen want and its neurologic implications.

Read before the Tri-State Assembly of Nurse Anesthetists, Chicago, April 28, 1952.

*Attending Neurosurgeon, Evangelical Deaconess and St. Luke's Hospitals.

Effects of Anesthetic Agents

The ability to produce voluntarily various degrees of depression of nervous function involves the production of a diminution in sensitivities of all types, motor as well as sensory. The basis of anesthesia is the fact that this decrease in sensitivities is variable, temporary, and reversible. The anesthetic agent has a selective effect on the phylogenetically more recent portions of the brain (the higher centers such as the cortex) and leaves the brain stem and its vital centers relatively free for the continuation of vegetative functions essential to the maintenance of life. The depression of the higher centers brings on progressive drowsiness and lethargy with an attendant decrease in and final-

ly alleviation of pain perception, with an accompanying depression of motor reactions, flaccidity, and unconsciousness. The anesthetic agent, whether it be a barbiturate administered parenterally or a gaseous anesthetic agent administered via the respiratory system, is carried by the blood stream to the brain, where by virtue of the lipid solubility, the lowering of surface tension, and the diminution in cellular permeability, it passes through the membranes into the brain cell proper. There is much current evidence to believe that an anesthetic agent depresses metabolism and thereby progressively decreases oxygen consumption, rather than acting as a cell toxin. Removal of the drug, as is done in normal metabolism and detoxication, reverses the influence on metabolism so that the oxygen consumption rises towards its original value. There is, furthermore, a selective differential gradient in the oxygen consumption of tissues, with brain metabolism being more sensitive than that of other tissues. However, it is necessary that adequate basic oxygenation be maintained constantly, inasmuch as oxygen represents the *sine qua non* of proper metabolism. Any decrease in the amount of necessary oxygen to below this critical level causes a profound alteration in nerve cells and frequently results in intractable cell destruction with its attending neurologic defects.

Types of Anoxia

The failure of an adequate oxygen supply to reach the tissues for any reason is variously called anoxia, oxygen lack, oxygen want, or hypoxia. Anoxia and hypoxia refer to the oxygen supply to the tissues, whereas anoxemia refers to low oxygen tensions in the blood stream. The various mechanisms of oxygen want may be listed as defective oxygenation of the blood in the lungs, lowered oxygen-carrying capacity of the blood, and slowing of the movement of the blood through the capillaries. These three mechanisms have given rise to the well know classification of Barcroft of the anoxic, anemic, and stagnant types of anoxia respectively. The anoxic type of anoxia may be produced by low oxygen tensions in inspired air, by abnormalities in the pulmonary mechanism, by pulmonary disease, by obstruction of the air passages, by paralysis of respiration, or by excessive pulmonary secretions. This type of anoxia is produced during anesthesia when there is an insufficient supply of oxygen, when there is obstruction of the airway for any reason, or when the patient drowns in his own vomitus or pulmonary secretions. The anemic type of anoxia is caused by hemorrhage and its attending anemia or by toxins causing anemia. The stagnant type of anoxia is due to slow circulation, such as is found in heart failure or profound shock. During anesthesia extreme blood loss can cause anemic anoxia, and prolonged shock can cause stagnant anoxia. It can readily be seen that during the course of a given anesthesia there is ample opportunity for the development of any one of the types of oxygen want.

A word about cyanosis in passing: The term refers merely to the purple, dusky, or blue discoloration of the skin and mucous membranes that is due to the presence in the superficial capil-

laries of reduced hemoglobin above a definite given amount. Inasmuch as such an increase in reduced hemoglobin reflects the lack of oxygen supply, cyanosis is interpreted as representing anoxia. However, oximeter readings have demonstrated that the oxygen tension of the blood is far below the critical level before cyanosis is present clinically; hence, cyanosis is a rather late sign of oxygen want.

Effects of Oxygen Want

The signs and symptoms of oxygen want depend on the rapidity of onset and the severity of oxygen lack. In slowly developing oxygen want the early changes may be so insignificant as to be overlooked, or a sudden decrease in blood pressure may be the first and only warning of slowly developing anoxia. At other times the symptoms may be an increase in respiratory effort, elevation in blood pressure and pulse rate, development of cardiac arrhythmias, muscular twitching, spasm, convulsions, extreme flaccidity, and finally death. The results of oxygen want depend on the severity and duration of the hypoxia and in part, perhaps, on the individual oxygen sensitivity of the patient. We have all heard of patients who have recovered from apparently severe states of hypoxia without obvious sequelae. It is well known, however, that severe states of anoxia over extended periods cause severe neurologic defects. Some of us have seen extensive pathologic changes and even death following severe and extreme instances of hypoxia. In determining the severity of brain injury in oxygen want, the important factors are the extent of nerve cell damage and the reversibility or irreversibility of the reaction. The clinical course following an episode of acute hypoxia varies. If the trauma to the brain cell has been minimal, apparently complete recovery without obvious or immediate sequelae may ensue. If the involvement has been extensive, the patient may have permanent neurologic residuals. In extreme cases death may ensue after a lucid interval or without the patient's regaining consciousness. Immediately following severe hypoxia respirations may be irregular, the body temperature is elevated, and there may be muscle twitches, convulsions, coma, and death. If death occurs within the first twenty-four to thirty-six hours, it is usually due to pulmonary edema, myocardial damage, and medullary failure. Patients who survive extreme hypoxic brain damage may be left with sequelae varying from mental and emotional abnormalities to Parkinsonism, generalized rigidity, dementia, and decerebrate states.

It is fairly well accepted that cerebral tissues are destroyed intractably when anoxia persists longer than seven to eight minutes. It is thought that various cerebral cell groups have varying thresholds of sensitivity to oxygen want, with some cells being more sensitive than others. With episodes of hypoxia of any extent or severity some cerebral cells are destroyed, regardless of whether the patient survives or dies, and regardless of whether he has obvious neurologic residuals. This neuronal destruction which occurs with hypoxia and anoxia and which is not necessarily associated

CENTRAL NERVOUS SYSTEM CELL DESTRUCTION VS FUNCTION

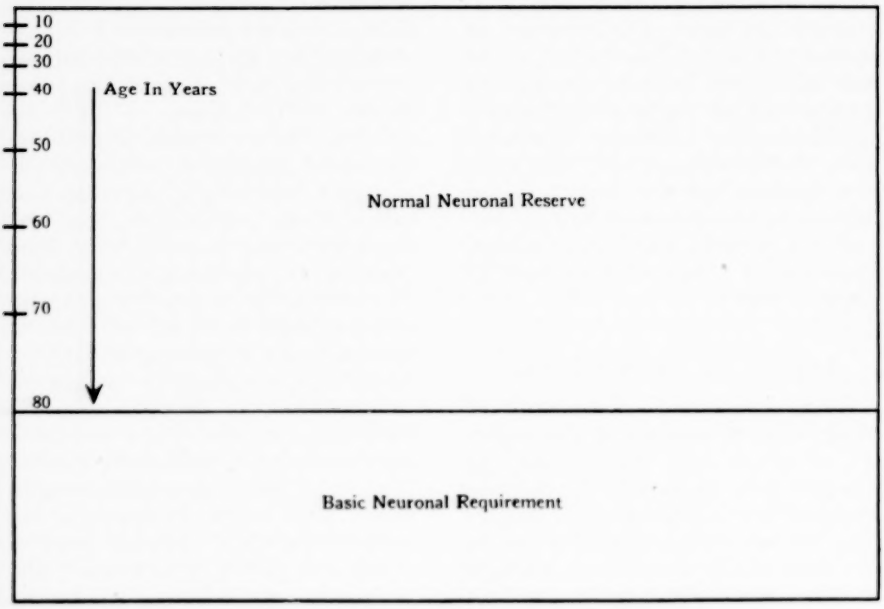

Fig. 1

with an obvious neurologic defect may be referred to as an insensible neuronal loss.

At birth the central nervous system is endowed with a multitude of neurons of various types serving a multitude of different functions. Many of these neurons subserve actual specific functions, such as motor power, sensibilities, speech, and vision, and may be collectively called the basic neuronal requirement. This group represents those cells whose destruction or alteration results in abnormalities of cerebral function or specific neurologic defects. The remainder of the cerebral cells, perhaps constituting a larger group than the former, is called the normal neuronal reserve (fig. 1). In these group differentiations the basic neuronal requirement may be thought of as consisting of those neurons that are physiologically active, and the normal neuronal reserve as consisting of those neurons that may be called upon in an emergency and that constitute the safety reservoir.

In the normal process of growing old the wear and tear of age on the central nervous system is manifested by a gradual reduction in the normal neuronal reserve, with eventual complete dissipation of the entire reserve occurring about the age of 70. With the depletion of the reserve and the beginning encroachment on the cells of the basic neuronal requirement, actual neurologic defects appear, such as loss of memory, forgetfulness, abnormalities in behavior, difficulty in coordination, tremor, convulsion, and dementia. The normal neuronal reserve, however, may be encroached upon

and even depleted prematurely by a multitude of insults to the central nervous system, such as trauma, infection, and anoxia. The summation of these insults added to the effects of age hastens depletion of the neuronal reserve. We may be completely unaware of this encroachment on the reserve, because it may occur as an insensible loss, usually following a specific insult to the central nervous system and not associated with any specific immediate neurologic defect. Nevertheless, the cell destruction and decrease in the normal neuronal reserve continue inexorably.

Some of the mechanisms causing cerebral damage are trauma due to birth or to external violence, acute infectious diseases with encephalitic involvement, and, perhaps the most important, anoxia from any cause, be it shock or oxygen deficiency during anesthesia. As a matter of fact, the mechanism of cell destruction in most of these insults is usually that of oxygen want. Whether the hypoxia be anoxic, anemic, or stagnant makes little difference. The resulting neuronal destruction is recorded in the neuronal ledger and subtracted, so to speak, from the neuronal balance. It is therefore apparent that the frequent recurrence of such anoxic insults to the central nervous system, whether resulting from trauma, infection, or lowered oxygen tensions during anesthesia, quickly adds up to the detriment of the patient. If and when the sum total of the incidents and the accompanying brain cell destruc-

CENTRAL NERVOUS SYSTEM CELL DESTRUCTION VS FUNCTION

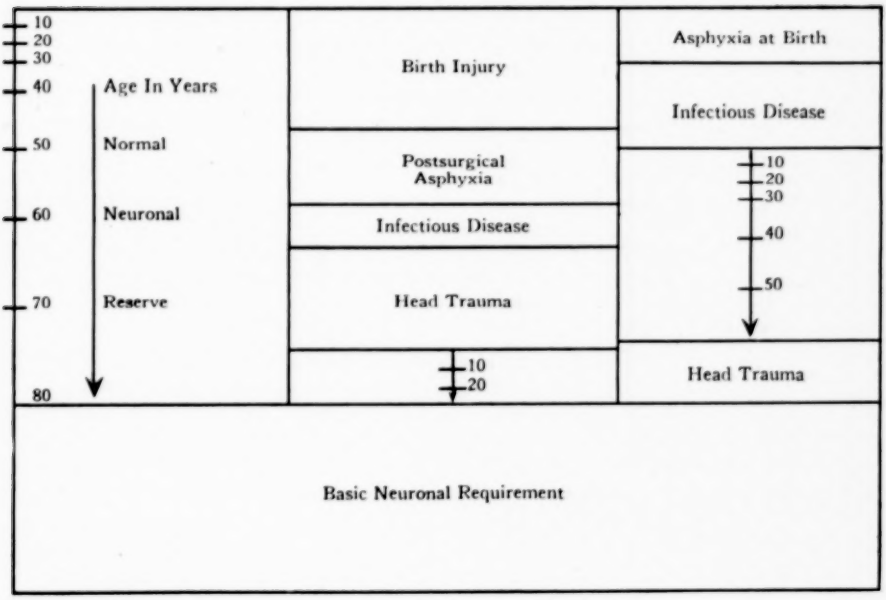

Fig. 2

tion, together with the insensible neuronal loss due to age, dissipates the entire reserve, actual encroachment on the basic neuronal requirement begins, and the individual has a specific neurologic defect. Even though the final episode precipitating the clinical neurologic defect may be relatively minor, it serves to catapult the patient into so-called cerebral decompensation (fig. 2).

It is important to realize that the tissue of the central nervous system is unusually sensitive to changes in oxygen tensions and to realize that most episodes of oxygen want are associated with some degree of persisting and intractable neuronal destruction. It is inconceivable that an infant should not have some degree of intractable neuronal destruction, even though insufficient to cause major neurologic residuals, when at birth he has respiratory center depression over an undue period, from excessive sedation of the mother during or immediately prior to delivery, and does not breathe spontaneously upon delivery but is finally resuscitated after some minutes to make what appears to be a satisfactory adjustment eventually. This loss, well termed an insensible neuronal loss—it may be compared to the insensible loss of water in respiration and perspiration—is nevertheless a specific and sizable loss, which cannot be set aside, for it is of sufficient caliber to be considered in the final accounting of the central nervous system reserve. Similarly, episodes of profound shock or persisting shock during a surgical procedure leave an indelible stamp on the nervous system structures in having helped to dissipate a portion of the normal neuronal reserve, in spite of the fact that the patient may not have an actual defect at the time. Acute infectious diseases, such as the infectious exanthems of childhood, are frequently associated with mild exacerbations and possibly fever and some confusion or delirium, all representing unrecognized encephalitic involvement, and similarly help deplete the neuronal reserve. In all of these various types of insult, even though apparently complete recovery takes place and the patient survives without an obvious defect, there is an appreciable insensible neuronal loss stamped on the neuronal reserve. When the summation of these various insults to the brain is sufficient to vault the barrier of the exhausted neuronal reserve, neurologic defects become apparent. At other times, the summation of destruction of neuronal reserve is merely added to that of the normal aging process, and months or years after an insult the person may pass into cerebral decompensation because of the premature dissipation of the neuronal reserve.

The ramifications of these thoughts on anoxic cell destruction are endless. There is some reason to believe, and supporting evidence to substantiate the belief, that so-called idiopathic epilepsy may be due to such insensible brain cell destruction early in life, with a decrease in the threshold of irritability for seizures or an exhaustion of the cerebral reserve for that particular neurologic reaction. Similarly, some of the chronic, progressive, demyelinizing diseases of the brain, such as multiple sclerosis and cortical atrophy, which have no obvious

(Continued on page 178)

Use of Depressant Drugs

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It seems expedient to approach the matter of the use of depressant drugs by listing a few of those drugs with their respective properties, the manner of their administration, adverse reactions to them, and applications appropriate to their properties.

In general, the drugs can be expected to provide analgesia, sedation or diminution of automatic nervous system activity. The most effective and safest use of the drugs can be anticipated if, preceding their administration, a reasonably precise evaluation of the needs of the patient is made and there is a working knowledge of the pharmacologic properties of the various drugs. In some instances pertinent effects can be secured with one drug. In other instances it is advisable to combine two or more drugs to obtain the desired effects with a minimum of undesirable side action. In respect to the use of combinations of drugs it is emphasized that specific indications must be established for each drug in the combination and that a "shotgun" type of therapy is not implied. If the physician is interested in the safety as well as the comfort of his patients, he can well afford to take the time necessary to learn the specific properties of old and new depressant drugs and make as accurate an evaluation as possible of the patient's particular need for the drug.

All too often sedative and analgesic drugs are given to patients without any consideration of the pharmacologic properties of the drugs, without anything more than a cursory evaluation of the dose required, and usually because in some prior, more or less similar situation the physician saw the drug given. To obtain a consistently safe and satisfactory response to sedative and analgesic drugs, it is expedient to use the drugs according to their respective pharmacologic effects and in a dose and by a route that will give reasonable assurance of a minimum of unnecessary depression and give the desired effect promptly.

The drugs to be discussed are representatives of two groups—the opiates and their substitutes and the barbiturates. In addition, alcohol administered intravenously has been used with some success to control pain in the postoperative period and as an adjunct to inhalation anesthesia.

Opiates and Their Subsititutes

Morphine

Properties.—Morphine possesses three significant properties useful

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in the areas previously outlined. It (1) reduces oxygen consumption, which is interpreted to mean a reduction in metabolic activity, (2) elevates the pain threshold, and (3) produces an appreciable diminution in the response to noxious stimuli. Morphine should be used in premedication and for all general purposes only when these properties are desired.

Route of administration.—Morphine may be administered by any one of three routes, depending on the time available for the production of its full effect. It may be given subcutaneously, and its full effect will be secured in approximately ninety minutes. It may be given intramuscularly, and its full effect will be obtained in approximately forty-five minutes. It may also be given intravenously, and its full effect will be manifest in approximately fifteen minutes. Administration of morphine by the intravenous route is an old established custom and a safe procedure, and it is unfortunate that use of this route of administration is not more widespread. It is useful not only in premedication for emergency operations but also for relief of the acute pain in such conditions as trauma and angina pectoris. The prompt action when morphine is given intravenously forestalls the tendency present when it is given subcutaneously to overdose the patient during the development of the effect of the drug.

Reactions and their treatment.—Unfavorable reactions to morphine are due most often to overdosage, which causes respiratory obstruction, depression, and asphyxia. The chief cause of hypoxia in morphine depression is not the reduction in respiratory rate but rather the respiratory obstruction that develops as the patient becomes narcotized and the jaw and tongue relax. If morphine depression is severe and prolonged, there also may be circulatory depression.

The treatment of morphine depression is aimed essentially at efficient oxygenation of the patient. The first step in treatment is establishment of the airway. Treatment of morphine depression or depression from any other drug will be frequently unsuccessful unless prompt and effective care is taken of the airway. After establishment of the airway, oxygen is given.

It must not be forgotten that, although oxygenation may be adequate in the severely depressed patient, there may be a high degree of carbon dioxide retention. Excessive retention of carbon dioxide may result in accentuation of the narcosis and deterioration of the circulation. It becomes necessary not only to guarantee oxygenation but also to provide sufficient pulmonary ventilation to eliminate carbon dioxide retention. The use of carbon dioxide inhalation is distinctly contraindicated in the treatment of morphine depression. It is not necessary nor rational to get the patient up and walk him about.

The use of analeptics such as coramine, metrazol, caffeine sodiobenzoate, alpha-lobeline, and benzedrine is of little value in the treatment of mild morphine depression and of no value in the treatment of severe morphine depression. It is dangerous practice to employ analeptics as the sole measure in the treatment of morphine depression in the fallacious belief that one is employing all

the treatment possible under the circumstances. The injudicious use of analeptics in the hypoxic patient may result in convulsions.

There are a number of substitutes for morphine that can be used with success and to advantage in patients who have a true idiosyncrasy to morphine. These are pantopon, dilaudid, demerol, methadon, and dromoran.

Pantopon

Properties.—Pantopon is a preparation containing many of the alkaloids of opium, including morphine in small amounts. It possesses the same properties as morphine and no significant additional properties.

Dilaudid

Properties.—Dilaudid is a synthetic analgesic drug possessing essentially the same properties as morphine.

Reactions and their treatment.—The significant advantage of dilaudid is its reduced tendency to produce nausea and emesis, and in this respect it is a satisfactory substitute for morphine in patients with a true idiosyncrasy. It is a somewhat more potent analgesic agent. It does, unfortunately, have a pronounced tendency to produce severe respiratory depression when given in amounts required to relieve pain, and this property is a potent deterrent to its common use as a morphine substitute.

dl-Methadon

Properties.—This synthetic preparation is capable of elevating the pain threshold and is somewhat more potent than morphine in this respect. It reduces metabolic activity but is less capable of influencing the response to noxious stimuli. It is this latter deficiency of the drug, as compared to morphine, that sometimes makes it an unsatisfactory substitute for morphine for the control of pain. With repeated doses there appears to be an accumulation of effect with the development of drowsiness.

Reactions and their treatment.—It is stated that there is less respiratory depression with the use of this preparation than with the use of morphine. In addition, less nausea and emesis seem to accompany its use. However, severe respiratory depression may occur; it is treated in the same manner as that accompanying morphine administration.

Demerol

Properties.—This is a synthetic preparation possessing essentially the same properties as morphine. However, it is less effective in reducing the response to noxious stimuli and therefore, like methadon, is not a satisfactory substitute for morphine for some patients with pain. It possesses an additional property of an atropine-like action. It is approximately one tenth as potent an analgesic as morphine.

Reactions and their treatment.—It is freely stated that there is less respiratory depression with the use of demerol than with the use of morphine. It may be that respiratory depression in the majority of patients is less pronounced, but it cannot be overlooked that demerol given in the usual therapeutic doses is capable of producing severe degrees of respiratory depression. Appreciably less nausea and emesis accompany the use of demerol than the use of morphine. It is not uncommon to note minor degrees of allergic responses to demerol, manifested usually by wheals at the site of

injection or along the course of veins after intravenous injection. No generalized allergic reactions have been observed, and no therapy for the minor reactions seems to have been necessary. However, there have been patients in whom either pronounced hypertension or hypotension appeared subsequent to the injection of demerol. The intravenous administration of doses larger than 50 mg. within fifteen minute periods often results in severe respiratory depression, although larger doses given by any other route or even at longer intervals intravenously are tolerated well.

Dromoran

Properties.—This synthetic preparation is capable of elevating the pain threshold to approximately the same extent as morphine. It is about twice as potent as morphine. It appears to reduce metabolic activity, but, like methadon, it has minimal influence on the response of patients to noxious stimuli. On repeated doses it also seems to produce drowsiness. The length of action is similar to that of morphine.

Barbiturates

These drugs are employed when there is need for (1) sedation (sleep or hypnosis), (2) some protection against the convulsive effects of cocaine and similar drugs, and (3) an anticonvulsant. It is well to remember that barbiturates are poor agents to use for pain relief and, when given in the presence of pain, often so influence the usual inhibitions controlling responses to noxious stimuli that a severe state of disorientation results, with exaggerated responses that are refractory. It is also well to remember that absolute protection against the convulsive manifestations of a reaction to cocaine and similar drugs is not guaranteed by the usual therapeutic dose of a barbiturate, and that an amount sufficient to produce almost complete hypnosis is needed to afford protection.

Drugs used.—The barbiturates are generally divided into four groups according to the length of action: an ultrashort-acting group, represented by pentothal sodium and evipal; a short-acting group, represented by such drugs as nembutal and seconal; a moderately long-acting group, represented by such drugs as amytal and dial; a long-acting group, represented by such drugs as luminal and veronal. The ultrashort-acting group acts for periods measured in minutes. The short-acting group exerts an influence for approximately four hours. The moderately long-acting group acts for about eight hours. The long-acting group is effective for twelve hours or longer.

Route of administration.—The barbiturates may be administered perorally, per rectum, intramuscularly, or intravenously. The route of administration depends to some extent on the availability of a soluble salt but chiefly on the desired time of onset of action and the length of action needed. Fortunately, preparations of all the groups are available for parenteral administration.

When drugs are given intravenously, the onset of action is short, and reasonably complete development of action can be expected from drugs of all groups within ten to fifteen minutes. The length of action is not significantly diminished if the drug is given

intravenously. This route permits administration of the drug in situations in which the obtaining of sedation and the control of convulsions are of an emergency nature. Furthermore, intravenous administration permits the full development of the effect of the drug when it is used for premedication and when the time before anesthesia is short, as is true in emergency operations. Also, it permits the use of the drug in patients to whom, for one reason or another, the drug cannot be given by mouth or rectum. Last, there is more constancy in the effect of the drug because there is less opportunity for alterations in absorption.

Reactions and their treatment.—Unfavorable reactions to the barbiturates are due most often to overdosage, causing respiratory depression, obstruction, and asphyxia. They often cause circulatory depression. The respiratory depression is characterized by shallow respirations at a normal or slightly increased rate, in contrast with the slow deep breathing of morphine depression. The treatment for barbiturate depression is essentially the same as that for morphine depression. The airway must be established, the patient must be oxygenated, and the circulatory depression relieved. The analeptic drugs have little or no value unless the depression is minimal, because the analeptics produce only brief stimulation, and the patient lapses again into the depression.

Alcohol

A low concentration of alcohol given intravenously has been demonstrated to be reasonably effective as an analgesic and also as a source of nutritional elements for patients requiring parenteral feeding. A satisfactory solution is one containing glucose, amino acids, and alcohol. The solution is prepared by adding 60-75 ml. of 95 per cent alcohol to a commercially prepared 1 L. bottle of aminosol (glucose and amino acids each in 5 per cent solution), proteolysate, or amigen. Depending on the caloric requirements of the patient, this amount plus an additional liter can be given in a twenty-four hour period, taking approximately four hours for each liter. Given at the rate of about 15 ml. per hour of alcohol, it will cause most patients to become mildly sedated and analgesic and not intoxicated. It cannot be expected that alcohol so administered will eliminate entirely the need for analgesic drugs in the postoperative period, but it may serve a useful function for many patients.

Alcohol given intravenously either as a complete anesthetic or as an adjunct to another type of anesthesia has been used erratically for many years. The results have not been uniformly satisfactory.

Dosage of Depressant Drugs

The dose of the drug to be given is influenced by a number of factors, including the potency of the drug, the physical state of the patient and his level of metabolic activity, the type of anesthesia to be used, the amount of drug previously given the patient, and the amount and type of drugs to be used in association with the drug. It is most important that the person ordering a drug of this

nature should avoid routine practices and adjust to each individual patient the dose required for that patient. Many of the undesirable responses associated with the use of depressant drugs are due to lack of attention to the individual needs of patients and not to the drugs themselves. There is an unfortunate tendency to give drugs on the basis of average tolerances, and there are too many patients who do not fit into the "average" category. Much more satisfactory results can be obtained by taking the time necessary to evaluate each patient and, in many situations, to give small repeated doses until the desired effect is secured. In addition, it is imperative that the person ordering drugs be familiar with their properties. In the case of new or unfamiliar preparations the physician is obligated to determine the properties from pharmacologic and clinical investigations. Ultimate determination of dosage depends on the individual's metabolic activity. Metabolism of the individual varies with his age. The most important single factor influencing the estimation of the level of reflex irritability, and therefore the tolerance to depressant drugs, is the patient's age. If anyone asked to prescribe a depressant drug were limited to one piece of information about the patient, the most valuable item would be the age. From a given value at birth, the metabolic activity increases sharply to 5 years of age, when it recedes slightly to rise again at puberty, after which it gradually declines until old age.

Basic levels of metabolism are increased by fever, pain, emotional disturbances, and specific hypermetabolic states (hyperthyroidism). Patients with such complications have a higher tolerance for depressant drugs and require larger doses than other patients.

Basic levels of metabolism are decreased in certain races (Negroes, Orientals) and by debilitating diseases, asthenia, and specific hypometabolic states (hypothyroidism). These persons are less tolerant of depressant drugs and require smaller doses than the average patient.

Postoperative Use of Depressant Drugs

There is reasonable evidence that the use of depressant drugs in the postoperative period contributes to the development of pulmonary inflammatory disorders and the development of phlebitis by limiting ventilation, diminishing the cough reflex, and reducing the patient's activity. However, patients are subject to pain and often require sedation, and drugs providing relief of this sort are needed. With sound application of the drugs it is possible to provide relief with a minimum of hazard.

It might be pertinent to the discussion of depressant drugs to report a few interesting observations made during a study of a comparison of the administration of dromoran and of morphine to 1,109 patients in the postoperative period.[1]

Eleven hundred and nine consecutive patients on the general and urologic surgical wards of the University of Iowa Hospitals were assigned to receive dromoran in 5 mg. doses or morphine in

1. Wilkins, D. S.; Cullen, S. C., and Brotman, M.: Intravenous supplementation during nitrous oxide anesthesia: Application of multiple covarient analysis to the comparison of demerol, morphine and a new potent analgesic drug (15431). Anesthesiology **12**:145-163, March 1951.

10 mg. doses subcutaneously for relief of pain in the postoperative period. Orders were written for the patients to receive only the designated drug every three hours pro re nata for pain. No other analgesic drugs were to be administered to the patients. Of the 1,005 patients who fulfilled the criteria for inclusion in the series, only 642 patients in the series received any analgesic drugs during the postoperative period. Because the drugs had been assigned preoperatively at random, the patients who received morphine totaled 338, and those who received dromoran totaled 304. Of those in the morphine group, 251 were given the full 10 mg. dose and 87 the 5 mg. dose. In the dromoran group 218 received the 5 mg. dose and 86 the 2.5 mg. dose. In all, the total number of doses of morphine was 1,117 and of dromoran, 1,036. The 642 patients who received either morphine or dromoran required an average of 3.4 doses each in the entire five day postoperative period.

The actual giving of the drugs was left to the discretion of the nursing staff, and the experiment represented the use of narcotics administered on that basis. The administration of 30.9 per cent of all doses was between the hours of 8 and 10 in the evening, and 8.9 per cent of the doses were given at precisely 9:00 p.m., which in this hospital is the time when the lights are turned out on the wards. It is possible, therefore, that a substantial portion of the drug doses was administered to patients who could not go to sleep and were prohibited from receiving hypnotics, which would have normally been given in those circumstances.

1. Only 64 per cent of the 1,005 patients required analgesia. The mean number of doses administered during the entire five days was 3.4.

2. A comparison of pain caused by various operative procedures was made, probably with fair validity. Patients who underwent deep thoracic, spinal column, and renal operations were the most outstanding in their requirements for analgesia.

3. A measure of the time course of postoperative pain was obtained, again with moderate validity. Patients who underwent spinal column and renal operations were outstanding in that their analgesic requirements did not greatly decline during the five day observation period.

4. An indication of the specific suitability of a given drug for relief of pain after a particular class of operation was found. For example, less morphine than dromoran was required by patients having spinal column operations, while the reverse was true of those having renal operations.

This brief presentation of a clinical study on analgesic drugs is included to given an example of the necessity for determining specificity of need for particular drugs. It also emphasizes the difficulties inherent in the clinical evaluation of drugs and the excellent opportunties for error in judgment.

Many patients exhibit pronounced restlessness, particularly in the immediate postoperative period. It is important to the patient's welfare that this restlessness be examined thoroughly with respect to the reasons for its development. The indiscriminate use of depressant drugs to control

restlessness too often results in undue depression. A frequent cause of restlessness is hypoxia. This possibility should not be overlooked, and oxygen should be given as a therapeutic test. Depressant drugs will enhance hypoxia because of hypoventilation and will control the restlessness only when given to the point of severe depression. Another cause of restlessness is response to pain during the stage of recovery from anesthesia, when there is a minimum of analgesia from the anesthetic agent but a retention of its influence on the cerebrum. In these circumstances the intravenous use of an analgesic in appropriate, and often small, doses will control the restlessness.

It is again emphasized that routine postoperative orders are less desirable than individualized single orders. Ideally, and particularly to patients whose tolerance to drugs is expected to be low, it is advisable to give small doses intravenously until the desired effect is secured. This is an especially advantageous procedure for the first and second doses and gives a reliable guide for subsequent doses. One will be regularly impressed with the relatively small dose required to control the distress. This will be especially true of patients whose overactive responses to noxious stimuli give an erroneous impression of the severity of the distress and lead the physician to order and administer unnecessarily large doses of depressant drugs.

Summary

An attempt has been made to outline some of the factors influencing the use of the depressant drugs. Although one can apply these drugs indiscriminately and achieve reasonably satisfactory results, it is felt that with rational application according to the properties of the drugs and the needs of the patient more satisfactory and safer results will be secured.

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Surital Sodium Anesthesia

Billie Caraway, R.N.*
Atlanta

Since 1949 a new intravenous anesthetic agent has been in use in the department of anesthesia at Georgia Baptist Hospital. This new anesthetic, surital® sodium, is a derivative of the barbiturates and a product of Parke, Davis and Company. Surital is the sodium salt of a thio-analogue of barbituric acid and is outstanding as a rapid, ultrashort-acting intravenous anesthetic. Its action is similar to that of pentothal sodium, although it is considered one and one-half times as potent as pentothal sodium. Procedures for administration of surital sodium do not differ materially from those for the administration of preparations of a similar nature. When surital sodium is properly used, its action is characterized by smooth, rapid induction and early, uncomplicated recovery for the patient. Dosage is adjusted according to response, and depth of anesthesia is determined by the common signs. In my experience, the dose of surital sodium required for induction is usually less than the dose of pentothal sodium, and the intervals between intermittent doses are much shorter, because detoxication of surital sodium is much more rapid. The cumulative action of surital sodium seems to be less than that of pentothal sodium. The channels by which surital sodium is detoxicated are essentially the same as those by which pentothal sodium is detoxicated.

The heart apparently is unaffected by surital sodium, since the pulse remains practically unchanged in rate and volume throughout operations uncomplicated by surgical shock. Respiration is somewhat more depressed than it is with the use of pentothal sodium, but a good depth can be maintained by giving sufficient oxygen. Induction of anesthesia with surital sodium is not attended by the sudden decrease in blood pressure that is sometimes encountered in induction with pentothal sodium.

Premedication for surital sodium anesthesia is essentially the same as that required for other anesthetic agents. In this series of cases any and all opiates were used with atropine sulfate or scopolamine. The preoperative hypodermic injection should be given at least one hour before operation.

In this series of 1,600 cases surital sodium-oxygen-curare was used in 900, and surital sodium supplemented with cyclopropane and nitrous oxide was used in the rest. Some spinal anesthesias were supplemented with surital sodium administered intermittently or by infusion.

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*Anesthetist, Georgia Baptist Hospital.

To determine exactly what could be done with surital sodium, we first used it without supplementary agents. Then gradually, but confidently, curare was added when relaxation was required. We were impressed with the number of patients who were awake, talking, and moving before leaving the operating room. Children, adults, and even aged patients tolerate surital sodium well. It is my impression that patients given surital sodium reacted with a clearer and more alert mind than those given pentothal sodium.

While it has been reported that surital sodium and pentothal sodium predispose to laryngospasm, in my opinion it is the manner in which the anesthetic is given that is at fault.

When indicated, an intratracheal tube is inserted to facilitate the performance of the operation. In many types of plastic operations around the face and head, multiple dental extractions, and tonsillectomies there is great benefit in the use of an intratracheal tube, especially if the patient has a short neck and good oxygenation is mandatory. Before intubation the patient's throat is well sprayed with a solution of cocaine, 10 per cent, or pontocaine, 2 per cent, at least fifteen minutes before anesthesia is begun. For some patients as many as three sprayings may be required. When the epiglottis may be elevated with the blade of the laryngoscope and the vocal cords sprayed without producing coughing, the throat is sufficiently anesthetized for the administration of anesthesia to be started. Surital sodium is given until the patient loses consciousness. Then 60 to 100 units of d-tubocurarine chloride is given intravenously. We use a 2.5 per cent solution of surital sodium in all cases. Within four or five minutes the jaw should be sufficiently relaxed to permit easy intubation. The administration of more surital sodium may be necessary at this time. Our method has been to use the dose necessary for the procedure.

Surital sodium gives an ideal anesthesia for radical mastoidectomy and fenestration operation on the ear. These are usually two to four hour procedures but do not require deep anesthesia. The local infiltration of novocain 1 per cent in these ear operations considerably reduces the dose of surital sodium required. We use surital sodium for all tonsillectomies in adults, a field which, in my opinion, requires great skill on the part of the anesthetist. The throat, of necessity, must be quiet despite the fact that stimulating suction, which could cause laryngospasm, is constantly being used. For many of our patients undergoing tonsillectomy intubation is not performed unless there is some doubt about the maintenance of a clear airway. However, these patients are prepared in the manner described regardless of whether intubation is done. Most of them are moving and talking before leaving the operating room.

Another type of operation that taxes the skill of the anesthetist is proctologic surgery. All of our patients undergoing rectal operations are placed in the jackknife position on the table before anesthesia, with both arms extended forward on armboards and with the head turned to one side. Induction with surital sodium should be rapid in these cases, and curare should be given immediately after

the patient loses consciousness. The position of the head is poor and laryngospasm is more likely to occur than with the patient in any other position. Again, an airway must not be inserted until the throat reflexes are abolished by curare. Oxygen is given through a catheter in the nose or through the airway.

In this series the following 911 operations were done with surital sodium-curare-oxygen anesthesia alone: plastic operations, 61; multiple dental extractions, 40; neurosurgical operations, 50; pelvic laparotomies, 95; upper abdominal operations, 75; proctologic operations, 170; genitourinary operations, 130; orthopedic operations, 60; ophthalmologic and otorhinolaryngologic operations, 130; gastroscopies, 100. The operations on the eye, ear, nose, and throat included bronchoscopies and esophagoscopies. The minimum dose of surital sodium used in the aforementioned procedures was 0.2 Gm., and the maximum dose was 3.6 Gm.

I have mentioned what I think are some of the most difficult types of operations that have been successfully performed with surital sodium anesthesia. Last, but not least, come the types of general surgery. We all know that technics of anesthesia for laparotomy may be simple or difficult. Many of our patients undergoing appendectomy, herniorrhaphy, gastric resection, or cholecystectomy with surital sodium-cyclopropane anesthesia reacted within twenty to thirty minutes after returning to bed.

The following case reports are representative:

CASE 1.—A woman, aged 42, was admitted for bilateral mammaplasty. Surital sodium-nitrous oxide-oxygen anesthesia was given. Time of operation was four hours and thirty minutes. Blood pressure remained at 120 mm. Hg systolic and 90 mm. Hg diastolic. Pulse rate was 80 to 92 a minute. Dose of surital sodium was 1.8 Gm. Dose of curare was 80 units. This patient was awake and talking before leaving the operating room.

CASE 2.—A woman, aged 22, was admitted for fenestration operation of the ear. The preoperative medication was morphine, gr. 1/6, atropine, gr. 1/150, and nembutal, gr. 1½, two hours before operation. After the anesthesia was started, morphine, gr. 1/6, was repeated. Surital sodium-oxygen-curare anesthesia was used, with the local infiltration of novocain 1 per cent in the ear. Blood pressure remained around 130 mm. Hg systolic and 80 mm. Hg diastolic. Pulse rate was 80 to 76 a minute. Respiratory rate was 20 a minute. Dose of surital sodium was 1.5 Gm. Dose of curare was 120 units given intermittently. Time of operation was four hours. The patient slept thirty minutes.

CASE 3.—A man, aged 44, was admitted with a gangrenous appendix. Length of operation was one hour and thirty minutes. Blood pressure was 140 mm. Hg systolic and 90 mm. Hg diastolic. Pulse rate was 96 a minute. Dose of surital sodium was 3.5 Gm. Dose of curare was 120 units. Patient moved ten minutes after being put to bed and talked thirty minutes later.

CASE 4.—A man was admitted for removal of bullet from his head. Preoperative medication consisted of morphine, gr. 1/3, and atropine, gr. 1/150. Blood pressure ranged from 100 mm. Hg systolic and 70 mm. Hg diastolic to 120 mm. Hg systolic and 80 mm. Hg diastolic. Pulse rate was 128 to 120 a minute. Surital sodium, 3.6 Gm., and curare, 120 units, were given. Operative time was five hours. Patient was returned to his room in good condition and was moving when put to bed.

CASE 5.—A woman, aged 48, was admitted for radical mastoidectomy. Preoperative medication consisted of morphine, gr. 1/6, and atropine, gr. 1/150, and the mediction was repeated thirty minutes after the operation began. Blood pressure ranged from 110 mm. Hg systolic and 80 mm. Hg diastolic to 120 mm. Hg systolic and 90 mm. Hg diastolic. Pulse rate was 100 to 90 a minute. Dose of surital sodium was 2.3 Gm., and of curare, 40 units. Operative procedure lasted three hours and twenty minutes. Patient was put to bed in good condition and was moving and groaning.

CASE 6.—A man, aged 57, was admitted for laryngectomy. Preoperative medication consisted of morphine, gr. 1/6, and atropine, gr. 1/150. The dose of morphine, gr. 1/6, was repeated thirty minutes after the operation started. Blood pressure ranged from 140 mm. Hg systolic and 90 mm. Hg diastolic to 170 mm. Hg systolic and 100 mm. Hg diastolic. Pulse rate was 104 to 90 a minute. Surital sodium, 2.7 Gm., and curare, 80 units, were given. Length of operation was three hours and forty-five minutes. Patient moved and opened his eyes when put to bed.

These cases were selected because of the length of the procedures and also because the dose of surital sodium was more than the average dose usually given. All of our patients undergoing major operations receive intravenous infusions of distilled water and dextrose 5 per cent or, when necessary, a transfusion.

SUMMARY

After reviewing 1,600 cases of unselected patients receiving surital sodium alone and in conjunction with curare, cyclopropane, and nitrous oxide for representative types of operations, we are convinced that it has a useful place in every department of anesthesia in which the anesthetists are trained in intravenous anesthesia.

Surital sodium is a safe anesthetic agent when properly administered. It is pleasant for the patient to take, and the reaction period is short.

In this series no nausea was encountered in patients receiving surital sodium alone. Laryngospasm occurred in a few instances, but no resuscitation was necessary, and no fatalities occurred.

HYPOXIA

(Continued from page 166)

specific etiologic background, may be due to such an exhaustion of the cerebral reserve.

SUMMARY

Oxygen want of any degree is capable of destroying cerebral cells. Repeated insults to the brain add up to the detriment of the patient. Episodes of oxygen want may be inadvertent, but the effect on the neuronal reserve is both intractable and, at times, disastrous. During anesthesia there is a multitude of opportunities for the development of hypoxia. Finally, anesthetists should remember the words of Haldane that oxygen want not only stops the machine but wrecks the machinery.

A.A.N.A.

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Use of Scanning Equipment to Observe Vital Physiologic Functions during Surgical Procedures

Henry A. Zimmerman, M.D. *
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Each year every large hospital has records of deaths from failure of the circulation during operative procedures. In one large general hospital seventeen cases of cardiac arrest occurred in an eight month period. In many of these cases the patient had no apparent cardiovascular disease and was considered a safe subject for surgery. In the last few years much has been accomplished in the field of cardiac resuscitation, and through these efforts the number of so-called cardiac arrests has decreased. Claude Beck, who pioneered in this field, has made available a course in cardiac resuscitation under the auspices of the Cleveland Heart Society.

In spite of improved methods of resuscitation many patients die because their condition is not noticed soon enough for resuscitation to be used effectively. For some years we have believed that if the physiologic functions of the patient could be more closely followed during the administration of anesthesia, many instances of cardiac arrest could be prevented. The methods used by the anesthetist to follow these functions during the induction phase are somewhat archaic, since it is physically impossible to record at this time the pulse, blood pressure, and amplitude phase and rate of respiration once or twice every five minutes. The anesthetist, however, is to be complimented on the skilful handling of patients in spite of these handicaps.

Several years ago we made a study on patients undergoing pneumonectomy to observe multiple physiologic phenomena at the time of the actual operation. The details of this study are reported elsewhere, but, in brief, we were able to record simultaneously the electrocardiogram, phase of respiration, right heart pressure, and femoral or brachial artery pressure by using strain gages, amplifiers, and recorded observation by direct writing equipment. During this study we became impressed with the fact that there are signs in most cases of cardiac arrest that forecast the standstill minutes before it actually occurs, and we believe that, if these signs can be detected, suitable measures can be taken to prevent the terminal event. The following three cases are cited to illustrate what can

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be accomplished with this type of observation.

CASE 1.—A man, aged 80, was undergoing right pneumonectomy for carcinoma of the lung. During induction of anesthesia it was noted that pulmonary artery pressure tended to increase steadily, and respiration quickened. Minutes before the onset of pulmonary edema we predicted its occurrence. Administration of the anesthetic was stopped, the patient was given oxygen under positive pressure, and the edema subsided. When the pulmonary artery pressure returned to a normal range, the patient was taken to the surgical ward. A week later the pneumonectomy was performed without complications.

CASE 2.—A man, aged 28, was undergoing pneumonectomy. During the induction of anesthesia first and second degree heart block developed in sequence, followed by complete dissociation within a matter of eight minutes. The ventricular rate then decreased rapidly until the patient had cardiac arrest. The operating surgeons reestablished circulation by cardiac massage, as they had been alerted to the possibility of cardiac arrest while they were still scrubbing, and were able to complete the pneumonectomy.

CASE 3.—A woman, aged 30, was undergoing pneumonectomy for a tuberculous cavity in the left lung. During dissection of the hilar area a supraventricular tachycardia with a rate of 180 a minute developed. This was not observed by the operating surgeon or his assistant. The anesthetist checked the temporal pulse and found the rate to be 90 a minute, only every other beat being palpable. Cedilanid, 1.6 mg., was given intravenously, and oxygen was administered. The rhythm returned to normal in ten minutes, and the operation was successfully completed.

Because of the impracticability of running yards of chart paper, we became interested in visual scanning equipment. We found that a scanner, the "Cardioscan," had been produced by the Sanborn Company but was no longer available as its manufacture had been discontinued. It consisted of a mirrored galvanometer, which projected the electrocardiogram onto a rotating drum. This was a satisfactory way of observing the electrocardiogram except that the observer had to peer through a small door to see the drum.

J. H. Rand III became interested in the problem and provided us with an industrial model of a cathode ray oscilloscope that had been used to record electrocardiograms. We were able to adapt the apparatus to blood pressure recordings, but we found that the scope was very sensitive and was always being influenced by stray 60 cycle currents, which could never be satisfactorily eliminated. Since then two models of this type have been produced commercially: one by the Carditron, and the other by the Cambridge Instrument Company.

Newman Fayerweather, an electronics instrument manufacturer, built us a custom model scanner, which we feel meets our requirements, as it is stable and durable, easily operated, and low priced. It is used for either electrocardiograms or blood pressure recordings, and it allows a complete visualization of these phenomena during the entire operative procedure. The scanner is so constructed that a direct writing recorder can be thrown into the circuit at any time, so that a permanent record of the data under observation can be secured.

The figure shows the assembled scanner. It consists of a mirrored pen motor with a light source that projects onto a calibrated revolving band. The band is so placed that it can be viewed by any number of persons.

Ideally, scanning equipment should have the following data available during the entire operative procedure: (1) electrocardiogram, (2) blood pressure from femoral or brachial artery, (3)

The compact scanner with one channel. Note the only control, which is the *off* and *on* switch. All other controls are from the electrocardiograph or the manometer, which makes for simplification of operation. The tape on the scanner moves at the same speed as the electrocardiograph's paper, i.e., 25 mm. per second.

phase of respiration, and (4) oxygen saturation of arterial blood by means of an oximeter with an ear attachment. Of the four the most important is probably the electrocardiogram, and we believe that with this alone a great deal could be accomplished towards preventing cardiac arrest during operations. It would appear that such a scanner could be made available in every operating room in the country for a nominal sum, and personnel could be trained in a short time to interpret the graph as obtained on the scanning equipment.

Summary

The problem of recognizing cardiac disorders during operations is discussed, and a method of scanning is described by which vital physiologic phenomena can be observed during the entire operative procedure. It is believed that many cardiac arrests in the operating room could be prevented with this type of equipment.

Transfusion Reactions

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One of the important advances in medicine in the last ten to fifteen years has been the increased availability and use of blood for transfusion. But like all good things in medicine, transfusions carry with them an element of danger. A definite morbidity and fatality rate attends their use.

The dangers of and difficulties from transfusions are of several different types with respect to etiology and also treatment, but they are collectively referred to as transfusion reactions. As a general statement, transfusion reactions are for the most part due to the faulty preparation or giving of blood, and the incidence of reactions is in inverse proportion to the care with which the blood is collected, stored, and given. Even with the best of care there is an incidence of 2 to 3 per cent of all types, most of them, fortunately, of the milder varieties. However, it is only by constant vigilance that such results are obtained, and the importance of unremitting watchfulness cannot be too highly emphasized. The more serious (and often fatal) reactions are distressing situations, and none the less so because many of them are avoidable.

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Anesthetists are likely to see the more severe forms of transfusion reactions, for it is generally true that the earlier in a transfusion the reaction occurs, the more severe it is. It is therefore incumbent on the anesthetist to be on the watch for untoward symptoms of any type and to discontinue the administration of blood until the nature of the reaction can be determined. Continuing the administration of blood under such circumstances may well convert a mild reaction into a fatal one.

Pyrogenic Reactions

As the name implies, pyrogenic reactions are due to the presence of pyrogens in the blood and therefore the contamination of the blood during its preparation. The incidence is about 2 per cent of all transfusions. This is not a serious form of reaction and is characterized by fever of varying degrees and chills, which start during, or within an hour of completing, the transfusion and last from one half hour to four hours.

For the milder cases no treatment is necessary, although if the patient is conscious salicylates may be given. For the more severe forms with chills 10 cc. calcium gluconate (10 per cent) may be administered intravenously or

small doses of morphine injected hypodermically.

Urticaria

It is not known why hives result from transfusions, but they occur in ½ to 1 per cent of the cases and last from one to five hours. If treatment is necessary, adrenalin (1/1,000 solution), 0.3 to 0.5 cc., should be administered hypodermically, or, if the patient is conscious, one of the antihistaminic drugs should be given.

Air Embolism

Air embolism is very rare but may result from failure to clamp the tube when all the blood has run in, so that air is sucked into the vein. A few cubic centimeters will cause no trouble, but large amounts may cause obstruction to the blood flow in the right side of the heart. About all that can be done for air embolism is to turn the patient on his left side and hope. This position favors release of the obstruction.

The next two types of transfusion reaction are mentioned merely for the sake of completeness, for they are not ones that anesthetists will see.

Serum Sickness

Serum sickness is rare, nor is it known why it should occur with the transfusion of human blood. There is an incubation period of five to twelve days. Fever, chills, skin rash, and pain and swelling of the joints and muscles occur.

Homologous Serum Jaundice

Homologous serum jaundice is infectious hepatitis due to the presence of a virus in the donor, and its occurrence is the reason for not using pooled blood or plasma. At the present time it is impossible to kill the virus without destroying the blood. There is an incubation period of two months. There is no way of preventing homologous serum jaundice except the ineffectual one of not using blood from anyone recently affected with jaundice. This rules out only the more obvious source of the virus, not the more common "carrier."

The next two types of reaction are serious ones which the anesthetist must recognize and for which she must at least initiate treatment.

Overloading of the Circulation

The circulation is overloaded when blood is given faster than the heart can handle it, and here it is proper to mention the rate at which transfusions should be given.

Blood is given to combat only two things, first, shock and, secondly, anemia, which to anesthetists means bleeding. Thus, the greater either of these is, the faster the blood should be given. Naturally, therefore, the rate will vary considerably depending upon the requirements of the individual patient, but blood should not be given faster than is necessary under the circumstances.

In the gravity method of giving blood, about 20 cc. per minute is the maximum rate obtainable. But for all ordinary purposes, with average blood loss and no shock, a rate of 5 to 10 cc. per minute is adequate, the slower rate being preferable. When there is any

question of a weak or diseased heart, the rate should be reduced to 1 cc. or, at most, 2 cc. per minute.

On the other hand, when shock is present, speed in the administration of blood is of the utmost importance. The first 500 cc. can be given in as short a time as fifteen minutes, and a second unit, within the first half hour. This rate, of course, necessitates the use of a technic other than the gravity method, e.g., a syringe or pressure bottle.

When the systolic blood pressure reaches 90 mm. Hg, the rate is reduced to 10 to 20 cc. per minute, and when over 100 mm. Hg, it is reduced to 3 to 5 cc. per minute.

A systolic blood pressure below 85 mm. Hg presupposes a loss of 25 per cent of the blood, and therefore about 1,500 cc. of transfused blood will be required to replace the loss. A good response is indicated by a rise of 10 mm. Hg in systolic blood pressure per 500 cc. of blood.

The overloading of the circulation is an emergency which, if untreated, can even within a few minutes result in death. The symptoms may appear during the transfusion or within an hour of its completion. Engorgement of the veins occurs and may most easily be seen in the neck or face. Cyanosis appears, and the patient becomes dyspneic. As the condition progresses, loud rales can be heard, and there is copious bronchial secretion, which becomes frothy and bloody. Cardiac irregularities occur, and death due to cardiac standstill supervenes abruptly.

Treatment must be prompt and energetic.

First, the transfusion must be stopped. Second, blood pressure cuffs should be applied high on all four extremities to allow ingress but prevent egress of the blood. The cuffs should be inflated to a pressure just under the diastolic blood pressure, usually, between 50 and 70 mm. Hg. It has been estimated that by this means about 750 cc. of blood can be removed from the general circulation. The cuffs cannot safely be left on longer than twenty to thirty minutes. Consequently, should it be necessary to continue this treatment longer, the cuffs can be inflated alternately to allow each limb a ten or fifteen minute respite. Third, once the immediate danger has been controlled as described, phlebotomy should be performed.

Hemolytic Reactions

Hemolytic reactions are due to the use of incompatible blood and usually mean a failure in technic. The blood has been improperly typed or labeled, or the wrong blood has been given. Occasionally, such a reaction occurs in spite of all precautions, particularly in such diseases as leukemia and hemolytic anemia. The incompatible blood causes hemolysis with the release of free hemoglobin into the blood stream. This produces shock and lower nephron nephrosis, which is fatal in nearly 50 per cent of the cases.

To understand this type of transfusion reaction, it is necessary to know something of kidney function, for it is the kidney that is most damaged by the reaction. In health the glomerulus of the kidney is a simple filter, allowing the fluid part of the blood to pass

through. This filtrate proceeds down the kidney tubules, which at first are straight but then are thrown into a whorl-like structure, the convoluted tubules. These are the important parts of the kidney, for they have the power to reabsorb the parts of the filtrate the body needs and to reject the undesirable waste products. For example, the filtrate contains sugar and urea in the same concentration as the blood, but the convoluted tubules completely reabsorb the sugar and completely reject the urea. Water occupies an intermediate position, for while most of it is reabsorbed, sufficient is left to carry off the urea and other waste products. In hemolytic reactions the convoluted tubules are so damaged that they reabsorb the filtrate indiscriminately and more or less completely. If all the filtrate is reabsorbed, anuria results; if nearly all is reabsorbed, the result is oliguria. In either case uremia develops and causes the fatal outcome.

The locale of the kidney damage also explains the term "lower nephron nephrosis," for the kidney unit, the nephron, consists of the upper part (glomerulus) and the lower part (convoluted tubules), and it is only the latter part which is damaged.

The clinical course of a hemolytic reaction falls into three distinct stages, each with different symptoms and each with a quite different treatment.

Stage I starts during or shortly after the transfusion and lasts one to twenty-four hours. The earlier it occurs, the more severe will be the effect. If the patient is conscious, he will complain of pain in the back and legs and constricting tightness in the chest. In any case there will be fever, chills, flushing, rapid pulse and respiration, and *shock*. The first step in treatment is to discontinue the administration of the blood that is being given and then treat the shock. This means giving blood that is compatible or, if this is not immediately available, using plasma.

Stage II, the stage of anuria, lasts from one to fourteen days. If urine formation is not resumed in this time, death results. Usually, the patient feels quite well, but no urine is voided. Increasing uremia occurs, but the greatest damage and danger come from the retention of water and electrolytes, and treatment is mainly directed to not adding to this load. In treatment the total intake of fluids for twenty-four hours should be 1,000 cc. plus the volume of urinary output. The intake of electrolytes, mainly sodium and potassium, should be very low or none. The diet should be almost exclusively carbohydrate. It should be noted that fruit juices are not suitable, for they are high in potassium content. If the patient is nauseated, the aforementioned requirements can be met by giving 10 or 15 per cent glucose in water in the volume indicated. The artificial kidney has also been of help in this stage.

Stage III is the stage of diuresis. This is the stage of recovery, but treatment must be as vigorous as in either stage I or II, for death can still result if treatment is inadequate. The lower nephron, which has been reabsorbing everything, suddenly reverses itself and reabsorbs very little. There is therefore a great outpouring of urine. A patient who

has been excreting several hundred cubic centimeters of urine one day will put out 4,000 or 5,000 cc. the next day, with a great loss of electrolytes from the body. The treatment now consists of forcing fluids and electrolytes so that the intake at least keeps up with urinary output.

It cannot be overemphasized that hemolytic reactions are serious, and that prevention is the most important part of treatment. Check and recheck that the right type and the right bottle of blood is being given.

The Patient in Shock

The problem of a patient in shock during anesthesia can be a difficult one to handle. It requires nice judgment to determine the proper treatment to follow. As can be seen from the foregoing discussion, shock can be due to either of two causes: first, the trauma of, or hemorrhage during, the operation, and, second, a hemolytic reaction. The treatment of these two types of shock is diametrically opposed. In the former the transfusion must be pushed; in the latter it must be promptly discontinued.

Each case must, of course, be judged on its own merits. Some cases will obviously be due to trauma and bleeding, and a few may be just as obviously due to the administration of incompatible blood. However, most hemolytic reactions will not be clearly recognized as such at first. Therefore the only safe rule is to discontinue the administration of blood when shock is present and not obviously due to trauma or bleeding. The shock should then be treated with new, properly tested blood, or, if such blood is not immediately available, plasma should be given until suitable blood is obtained. By following this procedure, the hemolytic reaction will be minimized as much as possible, and the danger of shock averted.

Summary

Reactions can be a real and sometimes fatal danger to recipients of blood transfusions. The incidence of such reactions varies inversely with the care bestowed in preparing and giving the blood. Even with optimal care there is an incidence of 2 to 3 per cent. Fortunately, most of these are of the milder types.

To prevent the more serious types of reactions it is important for all concerned to use every precaution. But if they do occur, it is just as important that they be recognized and treatment started quickly.

A clinical description of, and the appropriate treatment for, each of the several different types of transfusion reactions are given.

Neurosurgical Methods for the Relief of Pain

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The treatment of pain in most instances is directed at finding and eliminating the cause of this symptom. In a large percentage of cases relief can be accomplished by specific treatment of the cause. In a smaller group, however, specific treatment of the cause is not possible nor successful, and the physician is left with a symptom that must be dealt with by various methods to give the patient relief. Depending upon the type and degree of pain these various methods include (1) the use of analgesics, sedatives, and antispasmodics, (2) use of hormones, (3) psychotherapy, (4) x-ray and radium therapy, and (5) temporary interruption of the nerves transmitting or responsible for the pain by nerve block with a local anesthetic or alcohol. When these methods fail or are not applicable, the pain is said to be intractable, and a more drastic procedure is necessary for its control.

The commonest cause of intractable pain is cancer that has become inoperable by adjacent or remote extension into pain sensitive structures. A lesser, but still significant, group is composed of patients with pain from chronic visceral disease, such as pancreatitis and tabetic crises, atypical facial neuralgia, postherpetic neuralgia, Paget's disease with nerve root compression, phantom limb pain, and many other conditions.

The surgical procedures at the disposal of the neurosurgeon consist of operations that must interrupt the anatomic pathways utilized in the transmission of the pain impulse from its point of origin to its area of perception in the brain. In order to understand these procedures better, a simple review of the anatomy of the pain pathways is in order.

Pain Pathways

The end organs, or receptors, for the sensation of pain are thought to be free, or naked, nerve endings found in skin, mucous membrane, walls of blood vessels, cornea, and so forth. Stimuli received by the receptors are transmitted by way of afferent fibers, located in the spinal nerves V, VII, IX, and X for the head, and in the sympathetic nerves for the thoracic and abdominal viscera. These fibers enter the posterior roots of the spinal cord, or sensory roots of the aforementioned cranial nerves, and synapse on the nerve cells in the gray matter of

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the descending nucleus of the trigeminal nerve in the medulla or in the substantia gelatinosa Rolandi of the spinal cord. Fibers of second order neurons from these gray masses cross for the most part to the opposite side and ascend in the anterolateral quadrant of the spinal cord, the lateral funiculus of the medulla, the lateral portion of the reticular formation of pons and midbrain to the thalamus. Here they terminate on third order neurons in the ventral nuclei of the thalamus. At this point the knowledge of the exact mechanism whereby pain is perceived and appreciated as such ends. Third order neurons from the sensory nuclei of the thalamus are known to project to postcentral gyrus of the cerebral cortex, but this cortex is not necessary for the appreciation of pain as a pure sensation, for this sensation in its pure form is not greatly altered even after removal of a cerebral hemisphere.[1] By way of internuclear connections with other thalamic and hypothalamic nuclei and thalamocortical and hypothalamocortical connections, a mechanism is provided whereby pain sensation may be integrated with other modalities of sensation and modified by previous experience and emotion to become a complex memory pattern rather than a simple sensation. Under these circumstances, pain with a considerable emotional component may not be relieved by operations on the anatomic pathways for pain, and it may be necessary to perform prefrontal lobotomy, which removes the emotional component or suffering without greatly altering the perception of pain.

1. Evans, J. P., cited by Walker, A. Earl: *Central Representation of Pain* (Baltimore: The Williams and Wilkins Co., 1943).

Interruption of Cutaneous Nerve Endings

Undercutting or excising skin will sometimes relieve the pain of postherpetic neuralgia[2] and may be tried in severe cases, especially those on the trunk where the skin graft will not be disfiguring.

Peripheral Neurectomy

Section of selected peripheral nerves is occasionally performed for various disorders. The obturator nerve may be sectioned with some degree of success for relief of painful disease of the hip joint.[3] The lateral femoral cutaneous nerve of thigh may be divided for meralgia paraesthetica.[4] The sensory nerves supplying the skin of the toes and feet may be sectioned or crushed to give relief of pain induced by the ischemic changes of occlusive peripheral vascular disease,[5] and occasionally peripheral branches of the trigeminal nerve are sectioned or avulsed for trigeminal neuralgia. Due consideration must be given to the fact that section of a peripheral sensory nerve interrupts all modalities of sensation including position, and that a limb entirely without position sense is a useless one.

Dorsal Rhizotomy

Section of the posterior roots is applicable to the relief of intrac-

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3. Camitz, H.: Die Deformierende Hüftgelenksarthritis und speziell ihre Behandlung. Acta orthop. Scandinav. **4**:193-213, 1933.

4. Schlesinger, H.: Die Meralgia paresthetica (Bernhardt'sche Sensibilitäts-störung) Neuralgie und Parästhesien im gebiete der Nerves cutaneus femoris externus und ihre Bedeutung für die Chirurgie. Zentralbl. f. Grenzgab. d. Med. u. Chir. **3**:241-246, 1900.

5. Silbert, S.: A new method for the treatment of thrombo-angiitis obliterans. J.A.M.A. **79**:1765-1766, 1922.

table pain involving the face, neck, and trunk and in rare instances the extremities. It was first suggested by Dana in 1886 (cited by Foerster[6]) and carried out by Abbe[7] and Bennett[8] in 1889. This is a major operative procedure requiring laminectomy or combined craniectomy and laminectomy, but in properly selected cases gratifying relief is often obtained. The commonest conditions requiring section of the posterior roots of the cranial nerves are trigeminal[9] and glossopharyngeal neuralgia[10] and carcinoma of the paranasal sinuses, nasopharynx, and tongue.[11] In intractable neuralgias the appropriate posterior root is sectioned, rendering its respective area of distribution anesthetic but giving complete relief of pain. With the malignant lesions, extension of the carcinoma to neighboring structures of the face and neck usually makes it necessary to section the posterior roots of cranial nerves V and IX as well as the posterior roots of the upper cervical nerves. This can all be accomplished with the same operative exposure.

Some of the commoner lesions requiring section of posterior roots from the trunk and extremities are metastatic carcinoma involving the vertebral column or thoracic wall, Paget's disease of the spine with nerve compression, certain infections, and post-traumatic inflammatory processes involving the nerve roots of the trunk. As a general rule, it is necessary to section all of the nerve roots coming from the painful area. As is true of peripheral nerve sections, one must keep in mind that the dorsal roots are made up of fibers carrying all modalities of sensation, and section is not applicable to the treatment of pain in the extremities. One exception to this is an occasional case of degenerated intervertebral disk, usually lumbar, which has been unsuccessfully treated by direct approach and is accompanied by unremitting pain. Here section of a single dorsal root is often a most gratifying procedure, and the loss of a single root to an extremity does not leave the patient with a disabling sensory loss, because the receptive sensory fields of the root above and below overlap sufficiently to cover the area of sensation lost by section of the root between.

Anterolateral Cordotomy

This procedure was based on an observation by Spiller[12] of a patient with a loss of pain and temperature sensation in the lower extremities who later was found to have two small tuberculomas, one in each anterior quadrant of the spinal cord. Several years later in 1911 he[13] persuaded a surgeon by

6. Foerster, O.: Resection of the posterior spinal nerve roots in the treatment of gastric crises and spastic paralysis. Proc. Roy. Soc. Med. **4**:226-246, 1911.

7. Abbe, R.: A contribution to the surgery of the spine. M. Rec. **35**:149-152, 1889.

8. Bennett, W. H.: Paper given at Royal Medical and Chirurgical Society, April 23, 1889, Lancet **1**:839-840, 1889.

9. Spiller, W. G., and Frazier, C. H.: The division of the sensory root of the trigeminus for the relief of tic douloureux; an experimental, pathological, and clinical study, with a preliminary report of one surgically successful case. Philadelphia M. J. **8**:1039-1049, 1901.

10. Dandy, W.: Glossopharyngeal neuralgia (tic douloureux); its diagnosis and treatment. Arch. Surg. **15**:198-214, 1927.

11. Fay, T.: Intracranial division of the glossopharyngeal nerve combined with cervical rhizotomy for pain in inoperable carcinoma of the throat. Am. Surg. **84**:456-459, 1926.

12. Spiller, W. G.: The location within the spinal cord of the fibers for pain and temperature sensations. J. Nerv. & Ment. Dis. **32**:318-320, 1905.

13. Spiller, W. G., and Martin, E.: The treatment of persistent pain of organic origin in the lower part of the body by division of the anterolateral column of the spinal cord. J.A.M.A. **58**:1489-1490, 1912.

the name of Martin to section the anterolateral tract for relief of pain. This operation is ideally adapted to the relief of pain in one or both lower extremities or pelvis in patients who have a relatively long life expectancy. If the section is properly made, only the sensations of pain and temperature are lost. Section of the anterolateral tract will also relieve pain of visceral origin if a bilateral section is made. Disadvantages of the operation are transitory difficulty of bladder control with retention, due presumably to interruption of autonomic pathways in the spinal cord, and failure to obtain a persisting high level of analgesia. This latter objection can usually be overcome by making the spinal cord section at a high level in the cervical cord. Pain below the level of the umbilicus can usually be satisfactorily relieved by this method.

Medullary Spinothalamic Tractotomy

Because of failure to obtain high levels of analgesia with cordotomy, Schwartz and O'Leary[14] in 1941 carried out section of the spinothalamic tract in the medulla. When performed properly, this operation gives a level of analgesia up to the second cervical dermatome on the opposite side of the body. For this reason it is the procedure of choice for inoperable carcinoma of the apex of the lung with extension into brachial plexus and other lesions causing intractable pain in the upper extremity. Since this section is made in proximity to the respiratory and vasomotor centers in the medulla, bilateral section of this tract carries considerable risk, although Crawford[15] believed it feasible. Still higher levels of analgesia may be obtained by section of the spinothalamic tract in the pons[16] or midbrain[17] or by coagulation of this tract in the midbrain or thalamus.[18] There has not been enough experience with these last procedures to evaluate properly their place in the treatment of intractable pain.

Prefrontal Lobotomy

In patients with a large emotional component to their pain, and in whom the distribution of pain is such that it cannot be cured by section of anatomic pathways carrying pain sensation, prefrontal lobotomy may be indicated. This operation consists of interruption of the fibers connecting the dorsomedial nucleus of the thalamus with the prefrontal cortex and was first described by Moniz.[19] Since these pathways are not part of the anatomic mechanism for the sensation of pain, section of them does not alter the perception of pain as such. It dampens the patient's awareness of pain and relieves the anxiety and tension which his disease, and the pain consequent thereto, have produced. In other words, it re-

14. Schwartz, H. G., and O'Leary, J. L.: Section of the spinothalamic tract in the medulla with observations on the pathways for pain. Surgery **9**:183-193, 1941.

15. Crawford, A. S.: Medullary tractotomy for relief of intractable pain in upper levels. Arch. Surg. **55**:523-529, 1947.

16. Dogliotti, M.: First surgical sections, in man, of the lemniscus lateralis (pain-temperature path) at brain stem, for treatment of diffused rebellious pain. Anesth. & Analg. **17**:143-145, 1938.

17. Walker, A. E.: Mesencephalic tractotomy. A method for the relief of unilateral intractable pain. Arch. Surg. **44**:953-962, 1942.

18. Spiegel, E. A.; Wycis, H. T.; Freed, H., and Lee, A. J.: Stereoencephalotomy. Proc. Soc. Exper. Biol. & Med. **69**:175-177, 1948.

19. Moniz, E.: *Tentatives operatoires dans le traitement de certaines psychoses* (Paris: Masson & Cie, 1936).

lieves his suffering. Since this procedure has an effect on the personality, it should be used with caution and with the full understanding of the patient and his relatives. In my opinion this procedure is best used on patients with a large emotional component to their pain, when the life expectancy, owing to their disease, is relatively short, and when the pain cannot be satisfactorily handled in another manner.

Cortical Resection

In 1942 deGutiérrez-Mahoney[20] removed the representative portion of the postcentral gyrus of the cerebral cortex of a patient suffering from phantom limb pain. While subsequent follow-up studies have revealed that this procedure alone is not sufficient for prolonged relief of pain, it does relieve the pain temporarily. I have performed resection of the precentral and postcentral gyrus on two occasions for relief of phantom limb pain incident to removal of the limb for malignant disease. In both patients the pain was relieved for the duration of their lives (six and three months respectively), and they were not handicapped more than temporarily because of the procedure. There is no alteration of personality following such a procedure, and hence it does not have the objections of prefrontal lobotomy.

20. de Gutiérrez-Mahoney, C. G.: The treatment of painful phantom limb by removal of post-central cortex. J. Neurosurg. 1:156-162, 1944.

Summary

The treatment of intractable pain is a difficult but large part of the practice of medicine. When more simple methods of treatment fail, pain can often be relieved by a properly selected surgical procedure directed either at the anatomic pathways for pain sensation or at the mechanism whereby pain is elaborated from a simple sensation into a more complex emotional experience.

A. A. N. A.

Nineteenth Annual Meeting

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Notes and Case Reports

Static Electricity in Hospital Operating Suites: Direct and Related Hazards and Pertinent Remedies.[1]—Static electricity is a major cause of fires and explosions in hospital operating rooms and associated anesthetizing locations. The equipment in operating suites of most hospitals today includes much electrically nonconductive material that readily acquires electrostatic charge through handling. Many floors are made of highly resistant materials that impede the flow of electricity and prevent proper neutralization of charges. As a result, sparks of ignitible energy often occur in the vicinity of explosive mixtures of combustible anesthetic gases or vapors. Sparks have occurred at the edge of a breathing mask when it was touched for adjustment by an attendant. It seems likely, from postexplosion inspection, that small spark discharges may have occurred occasionally at barrier points inside anesthesia machines when rubber bags or breathing tubes were handled or large high-potential charges developed nearby.

The literature of several decades attests the reality of the electrostatic hazard in anesthetizing locations and of the severity of many of the resulting explosions. Numerous recommendations have been made for removing or reducing this hazard.

In the recent survey of hospitals by the authors of this paper, the abundant occurrence of static electricity in operating suites was clearly proved, and the efficacy of such recommended measures as could be applied was tested. Laboratory studies confirmed some observations. The numerous test details in this report are given to show the real magnitude of the electrostatic problem and to give every hospital some understanding of what its own hazards may be. The authors hope that the facts and considerations presented here will stimulate an interest in hospital circles for independent study and research and that eventually it can be said that there is probably no combination of equipment and personnel activity anywhere less liable to produce casual, dangerous charges of static electricity than that found in the anesthetizing areas of most hospitals.

In the light of this investigation, the following specific conclusions and recommendations appear to be important. The recommendations are minimal for the correction of static and other closely associated electrical hazards. They recognize the fact that many approved and desirable conductive materials and other items are not available at the present time, and that many hospitals cannot undertake the immediate reconstruction of all hazardous areas.

1. Guest, P. G.; Sikora, V. W., and Lewis, Bernard: Static electricity in hospital operating suites: direct and related hazards and pertinent remedies. Reprinted from Bureau of Mines Report of Investigations 4833.

Specific Conclusions

a. In the average operating suite there are many items of nonconductive material that singly or in combination permit and encourage the development of static electricity.

b. The presence of static charges can give rise to incendiary sparks and is a constant threat to patients and personnel in anesthetizing locations.

c. Many anesthetic gas mixtures, particularly those rich in oxygen, are highly explosive and easily ignited by static sparks. Some of the commonly used mixtures can be ignited by extremely low energy sparks of the order of 0.001 millijoule—less than a millionth of a gram-calorie.

d. There are a number of simple ways of preventing the development of static electricity in operating rooms, the most important being the substitution of articles of electrically conductive material for those of nonconductive, charge-producing material. Whether or not all of these substitutions are made, conductive floors and conductive contacts from equipment to floors should be provided to take care of any exigencies, such as the possible introduction into the operating room of some piece of unapproved material electrically charged or likely to become charged.

e. In operating suites that still have easily charged materials lying around, the quantities of induced charge apt to be produced and the potentials involved are such that neutralization can be brought about satisfactorily by conducting paths of fairly high resistance.

f. Floors and shoes of unnecessarily low resistance may give rise to spark and shock hazards in rooms where there are ordinary electrical appliances and portable lamps.

g. The use and proper installation in anesthetizing locations of electric lamps, motor, plugs, and receptacles that carry a third wire for positive grounding would reduce greatly the chance of personnel or patient receiving electric-power shock.

h. The correct amount of resistance in a static-charge neutralizing or ground circuit can be determined with adequate margins of safety from a knowledge of the rate of development of charge potential. Dangerous charges commonly require more than 0.1 second to develop.

i. As charge removal accompanies charge development on ground-connected personnel and equipment, conductive paths capable of removing or permitting neutralization of 99 per cent of any probable charge in 0.01 second will give a large practical margin of safety. Neutralization of *developing charges* on personnel or equipment in operating rooms or other anesthetizing locations can be adequately and safely accomplished in circuits of several megohms resistance.

j. Application of *preventive* measures must be thorough to assure complete removal of the static electricity hazard from anesthetizing locations.

Specific Recommendations

1. Wool blankets, plastic sheets, and most of the usual synthetic fabric materials should not be used around anesthetic gas equipment or patients anesthetized with combustible gas mix-

tures. Cotton blankets and sheets are satisfactory, but if these are stored in a warming compartment, some means should be provided to keep them from losing moisture. Before new types of materials are admitted to operating room or delivery room suites, they should be rigorously and competently tested under conditions of low relative humidity to be sure that they cannot readily develop high potential charge.

2. Conductive rubber mattresses, pads, and pillows should be substituted for the ordinary variety as soon as they are available. Conductive mattresses are strongly recommended for carriages, especially where nonconductive floors are still in use. Conductive covers are satisfactory if they completely and permanently enclose the nonconductive items. Conductive cover material is becoming available and should be put to use speedily.

3. Personnel wearing ordinary rubber or synthetic soled shoes may carry static, regardless of the type of floors. Conductive shoes, therefore, should be worn by all personnel who work around combustible anesthetics or anesthetized patients. Such shoes should have a resistance of not less than 0.1 megohm each, but they should not have or acquire an individual resistance of more than 0.5 to 1.0 megohm. Until approved conductive shoes can be obtained, leather shoes, unless they have rubber insoles or waterproofing, will usually suffice to bring about neutralization of body charges quickly. If the leather soles are reasonably conductive and recommendations 1, 5, 6, and 8 have been put into effect, the hazard introduced through the use of ordinary rubber or composition heels may be regarded as insignificant.

4. Suitable conductive rubber breathing tubes, masks, and bags should be installed on existing anesthesia machines as soon as they can be obtained; they should be standard equipment on all new machines.

5. Conductive floors should be used in all operating and delivery rooms and in other rooms where combustible anesthetics are administered. An effort should be made to keep floors within the limits of present code requirements and well above the lower limit. The opinion of the authors, based on many tests and considerations, is that the most satisfactory well seasoned floors will introduce in any neutralizing circuit or to the ground a resistance of not less than 0.2 megohm nor more than 2.0 megohms. Such floors will remove static charge adequately from any conductive bodies properly connected to them and prevent disconcerting, if not dangerous, shock due to possible defects in electrical equipment such as lamps and heaters.

6. Stools with smooth, rounded feet and bare metal tops are the most satisfactory ones electrically. If upholstered covers are needed for comfort or to prevent sliding or falling, they should be of conductive material. If stool tops are painted or enameled, these coatings should be removed. Wide cross straps of adhesive tape, sometimes used to prevent sliding, are not objectionable, but at least 50 per cent of the bare top surface of the stool seat should be left exposed.

7. Nonconductive casters, tires, and stool-leg tips should be replaced by conductive rubber or

other suitable material on all equipment used in the administration of combustible anesthetics or in the handling of anesthetized patients. If these cannot be obtained from the manufacturers of hospital supplies, wet towels to insure removal of static electricity from operating table, anesthesia machine, stool, and anesthetist should be used. Until it is possible for a hospital to install conductive floor in all hazardous areas, it is advisable to use wet towels in any instance, as they are much more effective in conducting electricity into floor materials of high resistance than any other method; wet towels can also be used effectively on carriages. If 1 megohm resistance-type intercouplers are employed, they must be kept in good repair and tested frequently.

8. Outer garments of wool, silk, or synthetic materials, such as rayon, nylon, and orlon, should be prohibited in anesthetizing locations. However, there seems to be no substantial evidence that personnel acquire static charge potentials as a result of wearing properly covered undergarments of these materials. Cotton uniforms are more suitable from an electrical standpoint than those of other materials because they retain a useful degree of conductivity at fairly low humidities and because they are not apt to produce or acquire charge by frictional contact with other cotton articles, such as sheets, bandages, and sterilized bundles.

9. Receptacles and plugs that cannot be pulled apart accidentally should be installed where needed. Unless they are of the explosion-proof type, they should not be placed lower than five feet above the floor or in any location where combustible anesthetic gases can pass over them or envelop them. Care should be taken to keep ordinary switches and open motors and heaters well out of range of combustible gas mixtures. *Electric wiring and equipment should be in conformity with the latest approved regulations of the National Fire Protection Association for hospital operating rooms wherever possible.*

10. A suitable measuring instrument should be installed in a convenient and safe place for testing the conductivity of shoes of all personnel entering areas where explosive gaseous anesthetics are administered. If conductive shoes cannot be obtained, shoes of leather or other absorbent material usually can be brought to a desirable value of conductivity in a few minutes by standing on a wet pad or piece of wet carpet. A little glycerin or hygroscopic salt, such as calcium chloride, in the water will give the treatment more permanence.

The authors believe that these recommendations for reducing to a minimum static-spark and ordinary electrical hazards in hospital anesthetizing areas are pertinent, practical, and essential.

The sixteenth qualifying examination for membership in the American Association of Nurse Anesthetists will be conducted on Saturday, Nov. 8, 1952.

The deadline for completed applications, including transcripts, is Oct. 3. If application with transcript is received too close to the deadline, the application may not be processed in time for the candidate to be scheduled for this examination. The decision of the Credentials Committee will not be mailed to candidates before Oct. 6.

Legislation

Child Awarded Heavy Damages for Administration of Undiluted Drug.[1]—A minor sought to recover the sum of $250,000 damages resulting from the alleged negligent injection by a United States Army doctor of concentrated Hartman-Ringer solution in the lower portion of her back.

Her mother was admitted to a Station Hospital as the dependent of her husband, an Army sergeant. The child was born on the day of admission. She was two months' premature. Five days after birth a nurse discovered that the infant was suffering from labored breathing and subsequently from alternate periods of no breathing with short breaths in between. The attending physician, an Army doctor, noticing the dehydration of the infant, decided that the injection was necessary to save the infant's life. The nurse then procured an ampule of the Hartman-Ringer solution, and the doctor administered 10 cc. Only then by reading the label on the ampule did he discover that the solution was in concentrated form and should have been diluted by one part solution to twenty parts of sterile water prior to the injection.

The child was awarded $94,650 in damages against the U. S. Government under the Federal Tort Claims Act, for the resulting injury to her spine, extensive scarring due to operations, and for future medical care.

1. United States of America v. Grigalauskas, 20 C.C.H. Neg. Cases 473-U.S.C.A.-1st; April 4, 1952.

No Damages Awarded for Mental Anguish in Sex Mixup Case.[2]—Both the attending physician and the hospital were sued for damages for the mental anguish of the parents alleged to have resulted from the fact that the plaintiffs were first informed that their child, which was born at the hospital, was a boy and later that it was a girl.

The records of the delivery room showed that the child was a boy, while the doctor stated that he was so busy that he could not remember the sex of the child.

There was no breach of contract, said the court, since there was no proof that the hospital did not deliver to the parents the child that was born to them. The misstatement with respect to the sex of the child was also not a breach of contract because the hospital was under no contractual duty to determine and report upon a fact so self evident. The law does not impose a duty to exercise care not to cause emotional disturbances. Hence the misstatements constituted no invasion of any legal rights of the parents. Before there can be a recovery even in the exceptional class of cases where such suffering is recognized as being the subject of compensation, it must be made to appear that it was clearly of a very serious nature. The court directed a verdict for the defendants.—**Emanuel Hayt, LL.B.**, Counsel for A.A.N.A.

2. McGahey v. Baptist Memorial Hospital, 20 C.C.H. Neg. Cases 605-Tenn.; March 10, 1952.

Nominations for Office American Association of Nurse Anesthetists

1952-53

PRESIDENT

Josephine Bunch (Shriners' Hospital for Crippled Children, Portland, Ore.): Graduate of Sacred Heart Hospital School of Nursing, Spokane, Wash.; graduate of St. Vincent's Hospital School of Anesthesia, Portland, Ore.; member of A.A.N.A. in good standing since 1936; former president, Oregon Association of Nurse Anesthetists; former chairman, Western States Assembly of Nurse Anesthetists; member, Board of Trustees, A.A.N.A., 1948-50; chairman, Personnel Practices Committee, A.A.N.A.; 1st vice president, A.A.N.A. 1950-52.

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Minnie V. Haas (St. Joseph's Hospital, Fort Worth, Tex.): Graduate of Methodist Deaconess Hospital School of Nursing, Rapid City, S. Dak.; graduate of Lakeside Hospital School of Anesthesia, Cleveland; member of A.A.N.A. in good standing since 1934; former president, Texas Association of Nurse Anesthetists; member, Board of Trustees, A.A.N.A., 1948-50; 2nd vice president, A.A.N.A., 1950-52.

Mary A. Costello (Cincinnati General Hospital, Cincinnati): Graduate of Mercy Hospital School of Nursing, Hamilton, Ohio; graduate of Cincinnati General Hospital School of Anesthesia, Cincinnati; member of A.A.-N.A. in good standing since 1943; former president, Ohio Association of Nurse Anesthetists; member, A.A.N.A. Board of Trustees, 1949-51; chairman, Educational Fund Committee, A.A.N.A.

2ND VICE PRESIDENT

Lillian G. Baird (University of Michigan Hospital, Ann Arbor): Graduate of University of Michigan School of Nursing; graduate of University of Michigan Hospital School of Anesthesia; member of A.A.N.A. in good standing since 1936; former president, Michigan Association of Nurse Anesthetists; member, Board of Trustees, A.A.N.A., 1949-51; member, Advisory to Approval Committee.

Edna Peterson (St. Francis Hospital, San Francisco): Graduate of St. Joseph's Hospital School of Nursing, Vancouver, Wash.; graduate of Sacred Heart Hospital School of Anesthesia, Spokane, Wash.; member of A.A.N.A. in good standing since 1941; former president, California Association of Nurse Anesthetists; former chairman, Western States Assembly of Nurse Anesthetists, chairman, Planning Committee, A.A.-N.A.; member, Board of Trustees, A.A.N.A., 1950-52.

Hazel J. Peterson (Deaconess Hospital, Minneapolis): Graduate of University of Minnesota School of Nursing, Minneapolis; graduate of St. Joseph's Hospital School of Anesthesia, Chicago; charter member, A.A.N.A.; former president, Minnesota Association of Nurse Anesthetists; former secretary, Minnesota Association of Nurse Anesthetists; chairman, Revisions Committee, A.A.N.A.; member, Board of Trustees, A.A.N.A., 1950-52.

TREASURER

Agnes M. Lange (Ravenswood Hospital, Chicago): Graduate of Mt. Carmel Hospital School of Nursing, Columbus, Ohio; graduate of St. Joseph's Hospital School of Anesthesia, Chicago; member of A.A.N.A. in good standing since 1935; secretary-treasurer, Indiana Association of Nurse Anesthetists, 1937-50; member, Tri-State Assembly Board of Trustees, 1940-50; treasurer, A.A.N.A., 1950-52.

TRUSTEES

Olive L. Berger (Johns Hopkins Hospital, Baltimore): Graduate of Roosevelt Hospital School of Nursing, New York City; graduate of the Johns Hopkins Hospital School of Anesthesia, Baltimore; member of A.A.N.A. in good standing since 1933; former president, Maryland Association of Nurse Anesthetists; former 2nd vice president, A.A.N.A.; former member, Educational and Nominating Committees, A.A.N.A.

Elizabeth Boyer (Veterans Administration Hospital, Cleveland): Graduate of Youngstown Hospital School of Nursing, Youngstown, Ohio; graduate of Jackson Park Hospital School of Anesthesia, Chicago; member of A.A.N.A. in good standing since 1934; former president and secretary-treasurer, Ohio Association of Nurse Anesthetists.

Clarene A. Carmichael (Norfolk General Hospital, Norfolk, Va.): Graduate of University of Missouri and University of Missouri School of Nursing, Columbia, Mo.; graduate of Johns Hopkins Hospital School of Anesthesia; member of A.A.N.A. in good standing since 1948.

Mary Copley (De Paul Hospital, Norfolk, Va.): Graduate of St. Vincent de Paul Hospital School of Nursing, Norfolk, Va.; graduate of St. Mary's Hospital School of Anesthesia, Detroit; member of A.A.N.A. in good standing since 1943; former vice president and president, Virginia Association of Nurse Anesthetists.

Rosella J. Crotty (Luther Hospital, Eau Claire, Wis.): Graduate of St. Mary's Hospital School of Nursing, Duluth; graduate of St. Mary's Hospital School of Anesthesia, Duluth; member of A.A.-N.A. in good standing since 1938; former president, Wisconsin Association of Nurse Anesthetists.

Pauline E. Henry (West Suburban Hospital, Oak Park, Ill.): Graduate of Washington Park Hospital School of Nursing, Chicago; graduate of Frances Willard Hospital School of Anesthesia, Chicago; member of A.A.N.A. in good standing since 1935; president, Illinois Association of Nurse Anesthetists; chairman, Tri-State Assembly of Nurse Anesthetists; member, Revisions Committee, A.A.N.A.

Madelyn Burns Knapp (Catonsville, Md.): Graduate of Memorial Hospital School of Nursing, Cumberland, Md.; graduate of Jewish Hospital School of Anesthesia, Philadelphia; member of A.A.-N.A. in good standing since 1947; former president, Maryland Association of Nurse Anesthetists.

Eva MacArthur (Brookline, Mass.): Graduate of Somerville Hospital School of Nursing, Somerville, Mass.; graduate of Lakeside School of Anesthesia, Cleveland; member of A.A.N.A. in good standing since 1933; former president, Massachusetts Association of Nurse Anesthetists; chairman, Northeastern Assembly of Nurse Anesthetists; former member, Nominating Committee, A.A.N.A.

Phyllis A. Roberts (Jefferson, Iowa): Graduate of Iowa Lutheran Hospital School of Nursing, Des Moines; graduate of University Hospitals School of Anesthesia, Cleveland; member of A.A.-N.A. in good standing since 1941; former president, Iowa Association of Nurse Anesthetists; secretary, Upper Midwest Conference of Nurse Anesthetists.

Mary H. Snively (Duke University Hospital, Durham, N.C.:) Graduate of Johns Hopkins Hospital School of Nursing; graduate of Johns Hopkins Hospital School of Anesthesia; member of A.A.-N.A. in good standing since 1934; former trustee, North Carolina Association of Nurse Anesthetists; former member, Publications and Examination Committees, A.A.N.A.; member, Curriculum Committee, A.A.N.A.

Lillian Stansfield (North Carolina Baptist Hospital, Winston-Salem): Graduate of Church and Home Infirmary School of Nursing, Baltimore; graduate of North Carolina Baptist Hospital School of Anesthesia; member of A.A.-N.A. in good standing since 1945; former president, Carolinas-Virginias Assembly of Nurse Anesthetists; vice president, North Carolina Association of Nurse Anesthetists.

Maudie Yonce (St. Vincent's Hospital, Jacksonville, Fla.): Graduate of Riverside Hospital School of Nursing, Jacksonville, Fla.; graduate of Charity Hospital School of Anesthesia, New Orleans; member of A.A.N.A. in good standing since 1947; former president, Florida Association of Nurse Anesthetists; former trustee, Southeastern Assembly of Nurse Anesthetists; member, Public Relations Committee, A.-A.N.A.

Alma D. Prykanoski (New Jersey State Hospital, Trenton, N. J.): Graduate of St. Francis Hospital School of Nursing, Trenton, N.J.; graduate of Mercy Hospital School of Anesthesia, Pittsburgh; member of A.A.N.A. in good standing since 1936; president, New Jersey Association of Nurse Anesthetists, 1950-53; trustee, Middle Atlantic Assembly of Nurse Anesthetists.

Nineteenth Annual Convention American Association of Nurse Anesthetists

September 15-18, 1952

PHILADELPHIA

Hotel Headquarters — Penn Sheraton

All General Sessions and the Business Session will be held in the Lecture Hall at Convention Hall.

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PROGRAM

Sunday, September 14

10 A.M.-6 P.M.—**Registration**

A.H.A. Headquarters—Bellevue Stratford

9:00 A.M.—**Assembly of Directors of Schools of Anesthesia**

Pennsylvania Room—Penn Sheraton

Bernard Bowen, R.N.
Chairman, Education Committee
Presiding Officer

Progress Report on A.A.N.A. *Curriculum Outline*

Helen Vos, R.N.
Director, Hurley Hospital School of Anesthesia
Flint, Mich.

2:00 P.M.—**Teaching Aids**

Bernard Bowen, R.N.
Director, Madison College Hospital School of Anesthesia
Madison College, Tenn.

Demonstration of Teaching Aids

Mary A. Costello, R.N.
Director, Cincinnati General Hospital School of Anesthesia
Cincinnati, Ohio

Monday, September 15

8:00 A.M.—**Registration**

A.H.A. Registration Desk—Convention Hall
A.A.N.A. Registration — A.A.N.A. Exhibit Booth (nos. 66-68)

9:00 A.M.—**Assembly of Directors of Schools of Anesthesia**
Lecture Hall, Convention Hall
Helen Lamb Powell, R.N.
Chairman, Advisory to Approval Committee
Presiding Officer

Report on Accreditation of Schools of Anesthesia
Adam R. Gilliland, Ph.D.
Professor of Psychology
Northwestern University, Evanston, Ill.
Cameron W. Meredith, Ph.D.
Assistant Professor of Education
Northwestern University, Evanston, Ill.

Pattern for Approval of Schools of Anesthesia
Margaret Sullivan, R.N.
Chairman, Approval Committee
Roosevelt Hospital, New York City

1:45 P.M.— **General Session**
Lecture Hall, Convention Hall
Verna E. Bean, R.N.
President, A.A.N.A.
Presiding Officer

Invocation
Sister M. Regina de Lourdes, R.N.
St. Agnes Hospital
Philadelphia

Address of Welcome from A.H.A.
Edwin L. Crosby, M.D.
President-elect, A.H.A.

Address of Welcome
Verna E. Bean, R.N.
President, A.A.N.A.
Lexington, Ky.

2:30 P.M.— Hilda Salomon, R.N.
Past President, A.A.N.A.
Presiding Officer

Forum: Reduction of Morbidity and Mortality in Obstetric Anesthesia

Introductory Remarks
Newlin F. Paxson, M.D.
Chief, Division of Women
Hahnemann Medical College and Hospital
Philadelphia

Intrauterine Studies
Franklin F. Snyder, M.D.
Assistant Professor of Obstetrics
Harvard Medical School
Boston

Cerebral Damage from Anoxia during Childbirth
Clement Smith, M.D.
Boston Lying-In Hospital
Boston

Discussion: Problems of Obstetric Anesthesia Service and How It May Be Improved
Newlin F. Paxson, M.D.
Chief, Division of Women
Hahnemann Medical College and Hospital
Philadelphia

Franklin F. Snyder, M.D.
Assistant Professor of Obstetrics
Harvard Medical School
Boston

Clement Smith, M.D.
Boston Lying-In Hospital
Boston

August Groeschel, M.D.
Assistant Administrator
New York Hospital
New York City

Victoria Scullen, R.N.
Obstetric Instructor
University of Pennsylvania Hospital
Philadelphia

Mary A. Costello, R.N.
Director, School of Anesthesia
Cincinnati General Hospital
Cincinnati

7:00 P.M.—**Friendship Dinner**
Della Robbia Room
Penn Sheraton Hotel

Tuesday, September 16

9:00 A.M.— **Business Session**
Lecture Hall, Convention Hall
Verna E. Bean, R.N.
President, A.A.N.A.
Presiding Officer

Call to Order
Report of Approval of Minutes Committee
Reports of Officers
Reports of Standing Committees

11:00 A.M.—**Election of Officers**

2:00 P.M.— **Business Session**
Lecture Hall, Convention Hall
Verna E. Bean, R.N.
President, A.A.N.A.
Presiding Officer

Reports of Standing Committees
Reports of Special Committees
Unfinished Business
New Business

Wednesday, September 17

Clinics at Philadelphia Hospitals
Lankenau Hospital
Children's Hospital
Graduate Hospital
Woman's Medical College Hospital
Jefferson Hospital
Methodist Episcopal Hospital
Mercy-Douglass Hospital
University of Pennsylvania Hospital
Temple Hospital
Bartin Memorial Hospital
Pennsylvania Hospital
Germantown Hospital
Episcopal Hospital
Jewish Hospital
Philadelphia General Hospital

9:00 A.M.— **Council Session**

Lecture Hall, Convention Hall
Florence A. McQuillen, R.N.
Executive Director, A.A.N.A.
Presiding Officer

2:00 P.M.— **General Session**

Lecture Hall, Convention Hall

Hazel Blanchard, R.N.
Past President, A.A.N.A.
Presiding Officer

Hospital Hazards and Their Control

George Thomas, M.D.
Chairman, Section on Anesthesiology
University of Pittsburgh
Pittsburgh

3:00 P.M.—**Anesthetic Agents with Physically Mediated Actions**

Joe B. Nash, Ph.D.
Research Associate
University of Texas Medical Branch
Galveston, Tex.

3:30 P.M.—**Electronically Controlled Anesthesia**

R. F. Courtin, M.D.
Section on Anesthesiology
Mayo Clinic
Rochester, Minn.

7:30 P.M.— **Banquet**

Ballroom, Penn Sheraton Hotel

Verna E. Bean, R.N.
President, A.A.N.A.
Presiding Officer

Invocation
Sister Marie Koch
Lutheran Deaconess Motherhouse
Philadelphia

Honoring of A.A.N.A. Past Presidents

Thursday, September 18

9:00 A.M.— **General Session**

Emma Miller, R.N.
Past President, Pennsylvania Association of
Nurse Anesthetists
Presiding Officer

9:00 A.M.—**Mechanism of Action of Volatile Anesthetics**

J. C. Krantz, Ph.D.
Professor of Pharmacology
University of Maryland School of Medicine
Baltimore

9:30 A.M.—**Control of Depth of Anesthesia**

William Hamelberg, M.D.
Department of Anesthesia
Ohio State University Hospital
Columbus, Ohio

10:00 A.M.—**Forum: Controversial Questions in Anesthesia**

Capt. Mary O'Carroll, ANC
Walter Reed Army Hospital, Washington, D.C.
Moderator

Controlled Respiration
Miriam G. Shupp, R.N.
University Hospitals of Cleveland
Cleveland

and

Hrant H. Stone, M.D.
Director, Department of Anesthesiology
Graduate Hospital
Philadelphia

Balanced Anesthesia

Helen Lamb Powell, R.N.
St. Louis

and

L. W. Krumperman, M.D.
Anesthesiologist, Temple University
Philadelphia

Atropine

Margherita Powers, R.N.
Johns Hopkins Hospital
Baltimore

and

Duncan A. Holaday, M.D.
Presbyterian Hospital
New York City

Respiratory Obstruction and Laryngospasm

Margaret Sullivan, R.N.
Roosevelt Hospital
New York City

and

George R. Brighton, M.D.
Attending Endoscopist, Roosevelt Hospital
New York City

Muscle Relaxants

Opal M. Schram, R.N.
Wesley Memorial Hospital
Chicago

and

Joseph F. Artusio, Jr., M.D.
Anesthetist in Charge, New York Hospital
New York City

2:00 P.M.— **General Session**

Problem Clinic

Minnie V. Haas, R.N.
1st Vice President, A.A.N.A.
Moderator

Alberta Boggan, R.N., Birmingham, Ala.
Frances Fanning, R.N., Ann Arbor, Mich.
Rosalie McDonald, R.N., Emory University, Ga.
Lucy Richards, R.N., Cleveland
Myra Van Arsdale, R.N., Cleveland

4:00 P.M.—**Unfinished Business**

Adjournment

Abstracts

Kolb, L. C.: Pain as a psychiatric problem. Journal-Lancet 72:50-54, Feb. 1952.

"The observations made on patients treated for intractable pain by prefrontal lobotomy have confirmed an impression long held in psychiatry that pain is not a simple percept but has significance as an emotional experience. . . . The prefrontal lobotomy has led to a separation of the emotional significance, the anguish or dread of future painful impressions, from the actual perception of the painful stimulus. It was pointed out many years ago that most hysterical pains have their origin in conjunction with an organically determined painful sensation to which the patient attaches by association a painful psychic impression which has occurred more or less simultaneously with the physical stimulus for pain. . . . It is not enough to state that pain may occur as an expression of an emotional disturbance or as a sign of anxiety. The question must be raised as to the manner in which pain may take on such significance. Briefly, the symptomatology of emotional disorders (neuroses or psychosis) is currently explained in the following manner. When the individual patient is confronted with some conflict between his inner drives and his personal and cultural standards which he is unable to resolve realistically, he may adopt (regress to) some form of childish or infantile behavior which in his fantasies gave him satisfaction in the past. . . .

"Pain as a complaint to solve a dependent need or to express anxiety associated with fears of self-mutilation through identification with another is most commonly encountered in persons of hysterical character. Masochistic self-punishment is a well-known psychiatric clinical phenomenon which underlies many chronic painful conditions. . . . No consideration of psychogenic pain is complete without mention of the complication of drug dependency or addiction. In addition to the psychologic needs of the patient expressed in his pain symptom, the long-continued use of a narcotic adds a physiological dependency through the development of addiction. With the occurrence of addiction the patient complains of pain in order to obtain the gratification provided by the drug and prevent the development of the unpleasant symptoms which occur with any abstinence from its use. . . . The psychiatrist is particularly interested in defects in the patient's social and emotional functioning as indications of stress and inability to adapt to his external environment. In the case of the patient's description of his painful illness the knowledge that some emotional disturbance is associated in time with the onset or recurrences of his painful complaint is suggestive that psychic processes are operative in its maintenance. . . . It

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Thoughts while holding up a chin—It's peculiar that the surgeon who is a slow operator, is so frequently also a late starter.

is important to determine the type of emotional disturbance from which the patient suffers, since the appropriate treatment for this condition will also prove the correct therapy for the painful symptom accompanying the illness. . . .

"In general, it may be expected that the patient's complaint is psychogenically determined when there are no signs of physical disease or the symptom has persisted long past the usual period of subsidence of a known acute illness or physical injury, when the symptom either is not relieved or is aggravated with the use of analgesics, when the symptom is relieved with the use of sedatives and hypnotics, when the patient appears indifferent to his symptom, when the pain is precipitated or aggravated by discussion of topics emotionally distressing to him, when it is relieved by intensive medical and nursing care which satisfied the patient's dependent needs, when the symptom is associated with character traits or symptoms indicative of a neurotic or psychotic disturbance, and when there is evidence from the patient's developmental history that he has undergone life experiences known to be productive of such illnesses. In contrast, patients with pain due to physical disease usually describe their symptoms precisely, signs of physical disease are commonly evident, analgesics provide either some or much relief, and the patient appears to be suffering and demonstrates a limitation of his actions consistent with the pain that he indicates he perceives. While such a patient may give a history suggestive of an emotional disturbance, more often such a history is not obtained. Early treatment of the painful complaint due to emotional disturbances is important. The earlier such treatment is instituted, the more likely that the condition may be modified by simple psychotherapeutic measures which do not have the disadvantage of the somatic psychiatric therapies such as electroshock and prefrontal lobotomy. Resolution of the painful symptom or its conversion into some other symptom is to be sought in all cases, since the patient with a chronic painful complaint is likely to be exposed to the danger of narcotic addiction and treated in such a manner that he may become totally dependent and disabled. . . .

"Although prefrontal lobotomy may be advised for the treatment of chronic painful conditions, it is now recognized that many patients make an unsatisfactory social adjustment following this procedure. . . . The patient whose pain complaint is symptomatic of an emotional disorder often presents a complicated diagnostic problem and commonly a major problem in therapeutic management. Remonstrating with such a patient or denying him the support of medication without providing him the insight and appropriate treatment for his psychiatric disorder usually leads to an accentuation of his symptom. In some instances drug dependency is the only satisfactory solution to the patient's dilemma."

HAMMES, E. M., JR.: Pain relieving drugs. Journal-Lancet 72:67-71, Feb. 1952.

"The complaints which lead a patient to consult his physician are many, but by all odds the commonest is a demand for the relief of pain or discomfort. . . .

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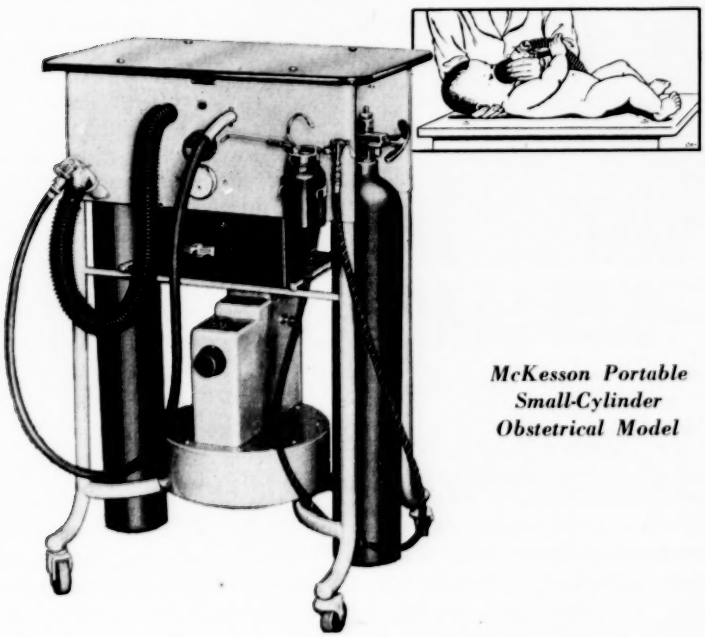

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Fortunately, the ability to relieve pain is among the greatest attributes of the physician. . . . Pain may be relieved in a great many ways: by mechanical means such as immobilization with splints or casts; by the use of heat or water to lessen spasm; by a surgical attack either on the source of pain or by surgically interrupting pain circuits in the peripheral or central nervous systems; by the use of anesthetic agents acting locally, in the spinal cord, or centrally on cerebral function; by psychotherapeutic attempts to distract from the awareness of pain or to increase individual tolerance of pain; by the use of drugs which reduce the pain-producing ability of a given disease process; and finally by means of analgesic drugs. . . . Broadly speaking, the drugs which relieve pain may be divided into four classes: (1) those which act on the organ or tissue producing the pain, such as cortisone in rheumatoid arthritis and ergot derivatives in vasodilatory headache; (2) those which act locally to block the conductivity of pain fibers, such as subcutaneous injection of procaine and the cutaneous application of ethyl chloride; (3) the true anesthetics which produce unconsciousness and thus relieve the awareness of pain, such as ether or the intravenous use of the barbiturates; and (4) the analgesics, which selectively depress the central pain-appreciation areas without loss of consciousness, such as aspirin and morphine. Of these groups, the first is the method of choice, since it most nearly approaches a rational attempt to restore normal function. . . .

"The commonly used and practically valuable analgesic drugs may be divided into two groups, depending on whether or not their repeated use can lead to addiction. Among the non-narcotic analgesics, the salicylates lead the list, headed by acetylsalicylic acid. . . . The paraminophenol analgesics consist of acetanilid and acetophenetidin (phenacetin). Acetanilid was first introduced in 1886, but early reports of toxic reactions led to a search for better tolerated analgesics in the same chemical group, and resulted in the development of the now more widely used acetophenetidin. The pyrazolon derivatives which include aminopyrine and antipyrine are mentioned only to condemn their use as clinical analgesics. The toxicity of these drugs in their ability to produce serious and occasionally fatal agranulocytosis is such as to make their clinical use unwarranted and foolhardy. . . . Among the narcotic analgesics, the opium alkaloids continue to enjoy the greatest use, and deservedly so. . . . The obvious limitations and undesirable side-effects of the opium alkaloids have resulted in a prolonged search, particularly in this country and in Germany, for better synthetic analgesics. . . .

"Among those synthetic analgesics which appear to deserve a place in present-day therapy may be mentioned the following: Meperidine (Demerol) . . . . Dihydromorphinone hydrochloride (Dilaudid). . . . [and] methadone hydrochloride (Dolophine). . . . The proper selection of the most effective analgesic, in any given circumstance, depends on the consideration of a number of factors. The first of these is an estimate of the severity of the pain. . . . The second consideration is

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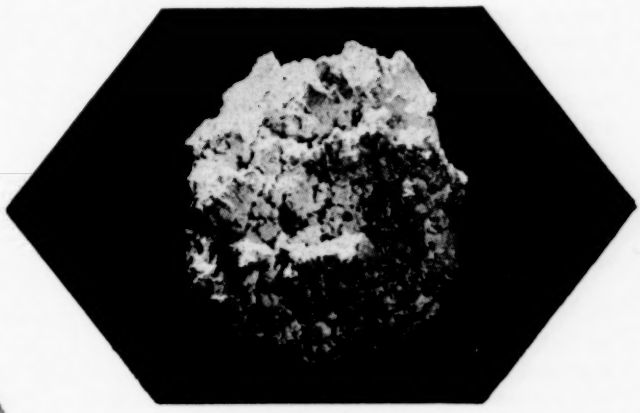

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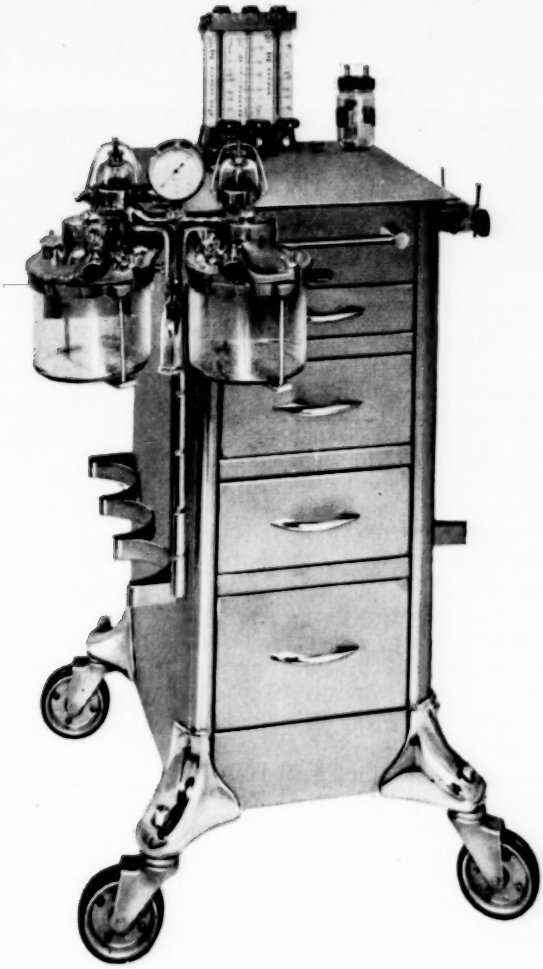
an evaluation of the side-effects and contraindications, including an awareness of the effect of the pain-producing illness on the excretion or destruction of the selected analgesic. . . . Thirdly, one must weigh carefully the purpose to which the analgesic is to be put and estimate the probable length of need."

Adelman, M. H.; Berman, R. A., and Touroff, A. S. W.: A new method of automatic controlled respiration. J. Thoracic Surg. **19**:817-820, May 1950.

"Controlled respiration may be defined as an anesthetic technique in which apnea is intentionally induced and respiration then maintained by intermittent inflation of the lungs. . . . The development of mechanical devices to maintain controlled respiration automatically was inevitable and the Scandinavian workers have been pre-eminent. . . . Mautz, one of the pioneers in our country, presented his experiences with a mechanical respirator for automatic and augmented breathing in 1941. In our approach to the problem of automatic controlled respiration, we held the belief that there was need for an apparatus less complicated than those described by the aforementioned workers. . . . The pneumatic balance resuscitator (PBR) is a light, simple, sturdy apparatus which permits of effective cleansing and sterilization. It was developed by Burns and studied extensively by Motley and co-workers. In brief, the PBR consists of a differential pressure valve which converts continuous positive pressure into intermittent positive pressure. Changes in positive pressure are obtained through a pressure-regulating device attached to the source of continuous pressure (oxygen cylinder).

"The differential pressure valve requires no adjustment. A nebulizer can be incorporated into the PBR for humidification of the dry oxygen gas and for the nebulization of antibiotics during anesthesia. We have used the pentothal-curare apneic technique in conjunction with the PBR in twenty cases. . . . The amount of Pentothal Sodium used in these cases was 0.85 to 1.5 Gm., while that of *d*-tubocurarine was 12 to 36 mg. We observed no fixed time requirement between the various doses of *d*-tubocurarine; in fact, there was considerable variation. Thus, a patient may remain in apnea as long as one and one-fourth hours before additional medication is required; this may be due, in part, to acapnia produced by the PBR which 'blows off' into the atmosphere on expiration. The duration of automatic controlled respiration in our cases ranged from forty-five minutes to three hours. It is of interest to note that, when spontaneous respirations reappear, a form of augmented respiration develops. This apparently is due to the fact that the differential pressure valve will cycle along with the respiratory pattern of the patient. Except for an initial fall in blood pressure following curarization, no untoward cardiovascular phenomena were observed; this fall in blood pressure was transitory. Oxygenation, as judged by the color of the blood, was good. Hilar manipulation did not initiate coughing or untoward reflex phenomena. Shifting of the mediastinum and paradoxical respiration were absent in the transthoracic cases. Operating conditions were excellent."



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Book Reviews

Anaesthetics and Anaesthesia for Nurses. By W. J. Finnie, M.B., Ch.B., B. Sc., D.A., Consulting Anaesthetist, Bradford Royal Infirmary, St. Luke's Hospital and Children's Hospital, Bradford, Examiner, General Nursing Council of England and Wales. Cloth. 110 pages, 36 illustrations. London: Nursing Mirror, 1951.

This small book, published in England for students and trained nurses, reflects the great difference in the systems of anesthesia service in Great Britain and the United States. The information presented is considerably more than is taught to the average American student nurse but infinitely less than is required of the nurse anesthetist.

In spite of this difference in application, nurses and nurse anesthetists in the United States will find much valuable information in the book. Nurses here will probably find the first chapter on premedication and preoperative care most useful. There is, however, a singular lack of instruction for the care of patients in the postanesthesia period.

The chapters pertaining to the anesthesia machines are of interest because common principles are involved. However, few British-made machines are used in this country.

Nurse anesthetists will probably find this book of most value in preparing lectures for student nurses.

Psychosomatic Gynecology. By William S. Kroger, M.D., Assistant Clinical Professor of Obstetrics and Gynecology, Chicago Medical School; Attending Obstetrician and Gynecologist, Edgewater Hospital, Chicago, and S. Charles Freed, M.D., Adjunct in Medicine, Mount Zion Hospital, San Francisco. Cloth. 503 pages. Philadelphia: W. B. Saunders Co., 1951.

Although this book is directed to the physician who treats gynecologic diseases, anesthetists will find of immediate interest chapter 6 on "suggestive relaxation," or hypnosis in labor, and the discussion of psychologic aspects of analgesia and anesthesia and of preoperative and postoperative care. The bibliography of seventy-five items following chapter 6 and of thirty-seven items following chapter 20 will be of added interest to anesthetists.

Medical Nursing. By Amy Frances Brown, R.N., M.S., Associate Professor of Medical Nursing, State University of Iowa College of Nursing; former Assistant Professor of Nursing, Frances Payne Bolton School of Nursing, Western Reserve University; Clinical Instructor and Supervisor in Medical Nursing, Medical College of Virginia School of Nursing; Instructor in Medical Nursing, Vanderbilt University. Ed. 2. Cloth. 1099 pages, 387 illustrations. Philadelphia: W. B. Saunders Co., 1952.

This comprehensive presentation of the subject of medical nursing contains many subjects that will be found of interest and value to nurse anesthetists. The chapters on nursing for diseases of the lungs have excellent presentations of the subjects of bronchoscopy, postural drainage, pulmonary edema, and other problems of importance in anesthesia. The chapter on nursing for heart disease has excellent charts and an outline of newer treatments for patent ductus arteriosus, tetralogy

of Fallot, and other conditions now being corrected by surgery.

Anesthetists will find innumerable instances of specific information, in addition to a general review of newer nursing procedures.

Early Care of the Seriously Wounded Man. By Henry K. Beecher, M.D., Chief, Department of Anesthesia, The Massachusetts General Hospital, Boston. Paper. 32 pages. Springfield, Ill.: Charles C Thomas, Publisher, 1952. $.75.

This paper-covered booklet presents the author's experience in wartime care of wounded men as projected into the civilian care of the wounded. The principles and methods of care are presented, from the problem of moving the wounded person to the time that the wound is repaired. Emphasis is placed on the importance of resuscitative procedures and on the dangers of overdosage with morphine. A concise summary of anesthesia for the wounded person completes the volume.

Principles and Practice of Anesthesiology. By Vincent J. Collins, M.D., Director, Department of Anesthesiology, St. Vincent's Hospital, New York City. Cloth. 528 pages, 99 illustrations. Philadelphia: Lea & Febiger, 1952. $10.00.

In the preface to this book the author says, "Knowing full well the many limitations that the work of a single individual on such a broad subject must necessarily have, . . . at the same time . . . it encompasses a total body of knowledge which has been considered to be in its broad outline the absolute minimum and necessary knowledge on the subject of Anesthesiology."

So broad a subject must of necessity result in a book that does cover a multitude of detail but leaves the reader with the wish that much more could have been said on many subjects. Reading through the book may at first fail to reveal the pattern of arrangement of chapters. It may be well to browse through the various chapters until the pattern becomes clear before attempting to use the book for study or reference.

The material is brought up to date and is presented on a case basis wherever that method of presentation is practicable. Six major areas of practice in anesthesiology comprise the sections into which the book is divided: general considerations, regional technics, pharmacologic considerations, complications, special considerations, and intravenous and inhalation therapy.

The scope of the material and the clinical approach will make this book valuable to the student of anesthesia.

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Classified Advertisements

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ANESTHETISTS: Urgently needed for 162 bed general hospital expanding to 247 bed. One hour from Pittsburgh. Maintenance, sick leave, holiday time, month vacation in addition to salary. Write: Administrator, Charleroi-Monessen Hospital, North Charleroi, Pa.; or telephone collect: Charleroi 3-5661.

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NURSE ANESTHETIST (A.A.N.A. member): 200 bed general hospital. To increase department now consisting of one anesthesiologist, four nurse anesthetists. Salary dependent on qualifications. Write Administrator, Samaritan Hospital, Troy, N. Y.

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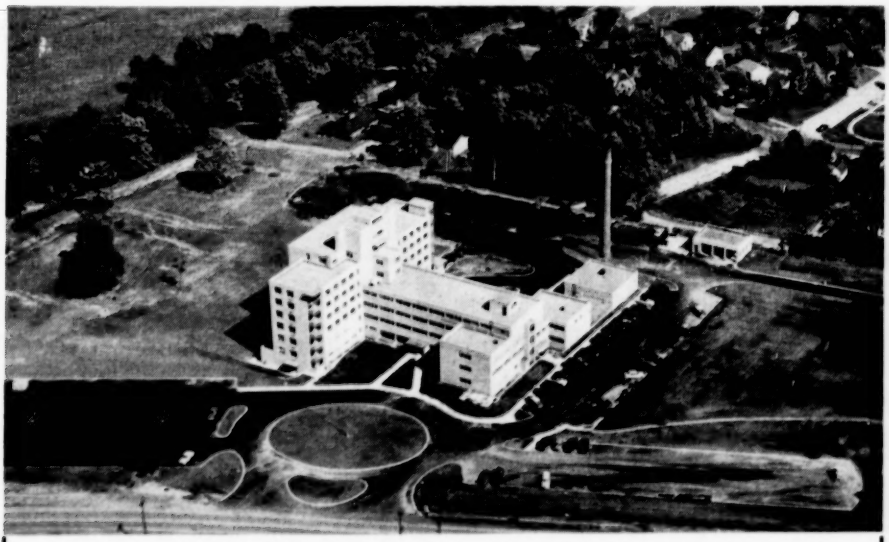

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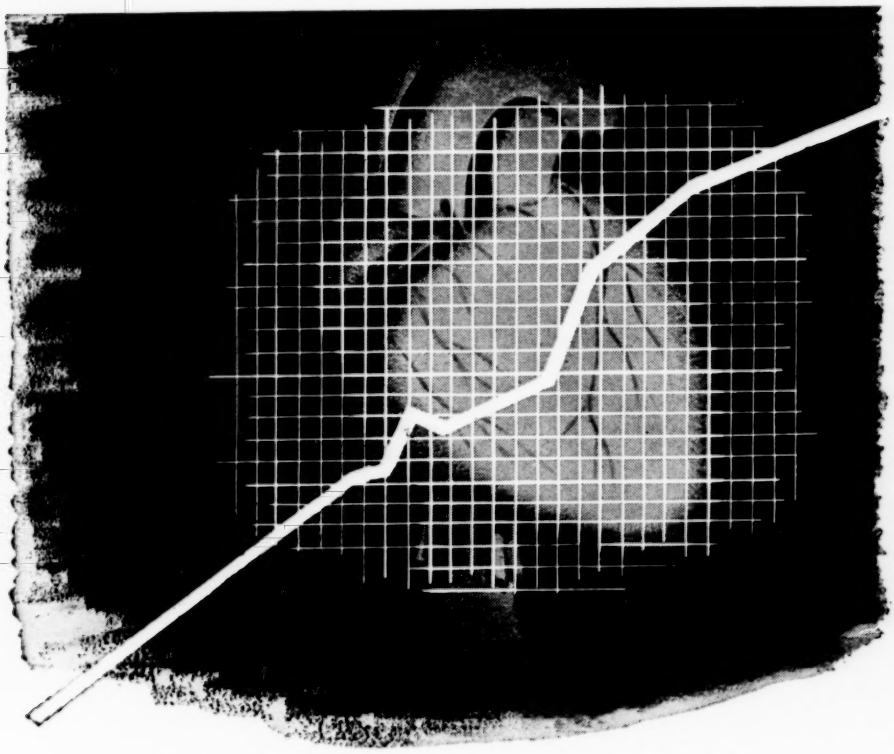

***Oxygen** has taken its place among the props for the failing heart*

"Oxygen Therapy in Heart Disease," a motion picture for physicians, has been produced by LINDE in collaboration with the American Heart Association, and has been accepted by the Committee on Medical Motion Pictures of the American College of Surgeons. Showings for medical groups can be arranged by calling or writing any LINDE office.

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